A STUDY ON ASSOCIATION BETWEEN BENIGN PROSTATIC HYPERPLASIA AND INGUINAL HERNIA

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CERTIFICATE

This is to certify that this dissertation titled **"A STUDY ON ASSOCIATION BETWEEN BENIGN PROSTATIC HYPERPLASIA AND INGUINAL HERNIA"** submitted by **DR. KAMAL RAJ. R** to the faculty of General Surgery, The TamilNadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MS degree Branch I General Surgery, is a bonafide research work carried out by him under our direct supervision and guidance from 2009 to 2011.

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CONTENTS

INTRODUCTION

Benign Prostatic Hyperplasia is an important cause of Bladder Outlet Obstruction in males leading to chronic straining on micturition. Chronic straining for micturition can precipitate inguinal hernia in adults[1,2]. The occurrence of inguinal hernia and benign hyperplasia with accompanied urinary tract obstructive symptoms are related to age. Benign obstructing prostate enlargement also predisposes to hernia and aggravation of symptoms related to hernia. Both Inguinal Hernia and symptomatic Benign Prostatic Hyperplasia are often found together in increased frequency in elderly [2, 3]. On the basis of this evidence significant correlation between inguinal hernia and obstructing benign prostate enlargement may be expected [2].

Several standard General Surgical text books give chronic straining due to BPH as an etiological factor for inguinal hernia in elderly male population[1,5,13]. But some of the studies showed that their occurrence together is considered a chance co-existence rather than cause and effect[3,14,15,16]. This study is aimed to find out whether Benign Prostatic Hyperplasia is a significant risk factor for developing Inguinal Hernia in males.

AIM OF THE STUDY

To find out the incidence of Benign Prostatic Hyperplasia among male patients with inguinal hernia.

To find out whether there is any causal association between Benign Prostatic Hyperplasia and Inguinal Hernia.

REVIEW OF LITERATURE

INGUINAL HERNIA

A hernia is a protrusion of a viscus or part of a viscus through an abnormal opening in the wall of its containing cavity [1].

The external abdominal hernia being the commonest variety, most frequent of it is the inguinal hernias. Important components of hernia are the hernia orifice and the hernia sac. Hernial orifice is the defect in the innermost apponeurotic layer of the abdominal wall and the hernia sac is the out pouching of the peritoneum. Neck of the sac corresponds to the hernial orifice.

INGUINAL HERNIA – ANATOMY [4, 5]

The groin region is a complex network of muscles, ligaments, and fascia that are interwoven in a complex fashion. Inguinal canal is 4 to 6cm long and is situated in the anteroinferior portion of the pelvic basin. Shaped like a cone, its base is at the superolateral margin of the basin, with its apex pointed inferomedially towards the symphysis pubis. The canal begins intra abdominally on the deep aspect of the abdominal wall, where the spermatic cord passes through a hiatus in the transeversalis fascia (in females, this is round ligament). This hiatus is termed the deep or internal inguinal ring. The canal ends at the superficial aspect of the abdominal musculature at the superficial ring, which is a defect in the external oblique apponeurosis.

Anteriorly the wall of the inguinal canal is formed by the external oblique apponeurosis and the internal oblique muscle laterally. Posteriorly, the wall is formed by the fusion of the transeversalis fascia and the transverses abdominus muscle, although up to one fourth of persons are found to have only the transeversalis fascia covering the posterior wall. Roof is formed by an arch formed by the fibres of the internal oblique muscle and the floor is formed by the inguinal ligament and the lacunar ligament.

The inguinal ligament is also known as the Paupart ligament and is comprised of the inferior fibres of the external oblique apponeurosis. The ligament stretches from the anterior superior iliac spine to the pubic tubercle. Cooper's ligament is otherwise known as the pectineal ligament. It is the lateral portion of the lacunar ligament that is fused to the periosteum of the pubic tubercle. The lacunar ligament, or the ligament of Gimbernat, is the triangular fanning out of the inguinal ligament as it joins the pubic tubercle.

Nerves of interest in the inguinal region are the ilioinguinal, iliohypogastric and genital branch of genitofemoral nerve. The ilioinguinal and iliohypogastric nerve arise together from first lumbar nerve (L1). The ilioinguinal nerve enters the inguinal canal by piercing the internal oblique muscle and exit through the superficial ring. The nerve supplies the skin of the upper and medial thigh and in males it also supplies the penis and upper scrotum, while supplying the mons pubis and labium majus in females. The iliohypogastric nerve arises from T12-L1 and

follows the ilioinguinal nerve. After the iliohypogastric nerve pierce the deep abdominal wall, it courses between the internal oblique and transeversus abdominis, supplying both. Then it branches in to a lateral and an anterior cutaneous branch, which pierces the internal oblique apponeurosis and then external oblique apponeurosis above the superficial inguinal ring. The Genitofemoral nerve arises from L1-L2, courses along the retroperitoneum, and emerges on the anterior aspect of the psoas. It then divides into genital and femoral branches. The genital branch enters the inguinal canal just lateral to inferior epigastric vessels. In males it travels through the superficial inguinal ring and supplies the scrotum and the cremaster muscle. In females, it supplies the mons pubis and labium majora.

Myopectineal Orifice of Fruchaud [4]: Fruchaud believed that all hernias of the groin begin within the groin; in an area he named the myopectineal orifice. This area in the groin is bounded superiorly by Arch of internal oblique muscle and transversus abdominis muscle, laterally by the Iliopsoas muscle, medially by lateral border of rectus muscle and its anterior lamina and inferiorly Pubic pectin. Running diagonally through the myopectineal orifice is the inguinal ligament. The orifice is covered by the transeversalis fascia.

INGUINAL HERNIA – EPIDEMIOLOGY [1, 5, 6, 7]

Inguinal hernia repair is one of the most commonly performed procedures by a General Surgeon. It is difficult to estimate the exact prevalence of inguinal hernia in the general population, but an overwhelming majority of the procedures are done in males 90% vs. 10% in females. Approximately 70% of femoral hernia repairs are done in females but inguinal hernia repairs are 5 times more common in females than femoral hernia repairs. Incidence of hernia have a bimodal distribution in males with peaks before 1 year of age and then again after 40 years of age. Indirect inguinal hernias are the most common hernias in both men and women; a right-sided predominance exists.

Exact prevalence of inguinal hernia in Indian population is not available. An extrapolated statistical analysis from a study given by Asia Pacific Hernia Society states that around 1,95,7850 people are affected by inguinal hernia in India[7].

INGUINAL HERNIA – ETIOLOGY

Inguinal hernias may be considered congenital or acquired diseases. A number of studies have attempted to delineate the precise cause of inguinal hernia formation; however the risk factors are likely multifactorial.

Presumed Causes of Groin Herniation[2, 3, 5, 13]

Coughing	Birth weight < 1500g
COPD	Positive family history
Obesity	Valsalva maneuver
Straining – constipation	Ascitis
Straining – prostatism	Upright posture
pregnancy	Connective tissue diseases
Defective collagen Synthesis	Previous Right Lower Quadrant incision
Arterial aneurysms	Cigarette smoking
Heavy lifting	Physical exertion

Congenital hernias are considered to be an impedance to the normal development, rather than acquired weakness. During the normal course of development, the testes descent from the intraabdominal space into the scrotum in the third trimester. Their descent is preceded by the gubernaculums and a diverticulum of peritoneum, which protrude through the inguinal canal and ultimately becomes the processus vaginalis. Processus vaginalis usually closes between 36 and 40 weeks, failure of which results in patent processus vaginalis and this explains the high incidence of indirect inguinal hernia in preterm babies.

The introduction of continuous ambulatory peritoneal dialysis for renal failure has demonstrated, as did ascites, that a patent processus vaginalis or canal of nuck, if subjected to increased intraabdominal pressure over a period of time, will dilate and produce a Hydrocele or hernia. Carcinomatosis, decompensated liver or heart disease can therefore present as recent onset herniation.

Microscopic examination of skin of inguinal hernia patients demonstrated significantly decreased ratios of collagen type I to collagen type III. Type III collagen have less tensile strength than type I collagen. Additional analysis of the skin revealed disaggregated collagen tracts with decreased collagen fibre density. These connective tissue defects are seen systemically – in skin, lung, pericardium etc. Most common cause for these changes is found to be heavy smoking and the condition is termed as metastatic emphysema. Therefore long term tobacco exposure is a risk for hernia formation. Patients with congenital connective tissue disorders like Ehler Danlos Syndrome, due to defective connective tissue, leading to less tensile strength of their tissues, can have hernia formation.

Historically, hernia causation was attributed to a mechanical disparity between visceral pressure and the resistance of the musculature. Cooper not only identified Transeversalis fascia, but also pointed out it was the last barrier to groin hernia formation. Therefore factors that increases intraabdominal pressure – cough, obesity, unusual exertion, pregnancy, prostatism in old age patients, over the long

time can lead on to hernia formation. However this occurs in a combination with a patent processus vaginalis or through age related weakness of the abdominal musculature is not clear. Both Inguinal Hernia and symptomatic Benign Prostatic Hyperplasia are often found together in increased frequency in elderly [2, 3]. On the basis of this evidence significant correlation between inguinal hernia and obstructing benign prostate enlargement may be expected.

PROSTATE – ANOTOMY[4, 9]

The prostate gland is formed around the end of the third month (first trimester) from the epithelium of the future prostatic urethra. The epithelium proliferates and penetrates the surrounding mesenchyme, which is the future fibro muscular prostatic tissue.

The classical description of the adult prostate is that it has the size, shape, and consistency of a large chestnut. The form of the prostate is that of a compressed inverted cone: pyramidal, having a base and an apex. It is located between the vesical neck of the bladder and the apex of the urogenital diaphragm. The normal weight of the prostate in a young adult is from 17 to 19g. The numbers 4, 3, 2 are useful as a mnemonic for remembering the transverse, vertical, and sagittal dimensions in centimetres, respectively, of the gland. The prostate is enveloped by extra peritoneal connective tissues that cover the thin anatomic

capsule (true capsule) of the organ, and it in turn envelops the proximal male urethra. The prostate is fixed to its location by Puboprostatic ligaments, Urogenital diaphragm, Bladder, Prostatic sheath and Fascia of Denonvilliers.

Prostatic Surfaces

There are four prostatic surfaces: one posterior, one anterior, and two inferolateral. The posterior surface is flat transversely and convex vertically. It is separated from the rectal ampulla by the bilaminar fascia of Denonvilliers. This surface is characterized by a midline groove that is wider toward the base of the gland, and serves to partially separate the gland posteriorly into left and right lobes. The posterior surface may be palpated by digital rectal examination. The vesicoprostatic junction is located at the upper border of the posterior surface. The narrow and convex anterior surface is located between the apex and the base. Multiple large veins separate this surface from the symphysis pubis. The avascular puboprostatic ligaments are fibrous cords, wide or narrow. They connect the upper limits of the anterior surface of the prostate to the pubic bone, at the right and left sides of the cartilaginous area. The right and left inferolateral surfaces are embraced by the anterior part of the levator ani muscles. They are fixed to the levator by the arcus tendineus of the fascia pelvis, sagittal connective tissue bands between the ischial spine, and the pubic bone. Here there is a very rich venous network and fibrous tissue which contributes part of the lateral prostatic sheath.

Prostatic Urethra

The prostatic urethra begins at the urethral meatus at the apex of the trigone of the bladder. This opening is crescent-shaped, invaginated posteriorly by a protuberance caused by the underlying glandular tissue (median lobe of the prostate), thus forming the uvula vesicae. This is continuous with a posterior midline urethral ridge, or crest, in the urethra. The urethral ridge has a distinctly expanded portion called the verumontanum, or seminal colliculus.

Prostate – Structure

McNeal described four regions or zones in the prostate: peripheral, central, transition, and anterior fibromuscular stroma. The urethra is the key anatomic entity defining these regions. Posterior to the urethra is the glandular area. Anterior to the urethra is the fibromuscular area; that is, the ventral portion of the glandular prostatic tissue is covered by the fibromuscular stroma.

Peripheral Zone: It is likely that the glands of this zone develop from the urogenital sinus and drain into the prostatic urethra. Nearly 75% of the glandular prostate, the peripheral zone surrounds most of the central zone and much of the urethra; in other words, it surrounds the posterior and lateral areas of the prostate gland. Its glands drain into the prostatic urethra. This zone is formed by multiple tubuloalveolar glands. The long, narrow ducts of this zone branch into small,

round, regular acini with smooth, nonseptate walls. Epithelium is simple columnar; its pale cells have distinct borders and basally-placed small, dark nuclei. Most carcinomas develop in the peripheral zone.

Central Zone: Ducts of this zone are probably of wolffian origin. The central zone, which is nearly 25% of the glandular prostatic parenchyma, envelops the ejaculatory ducts and extends toward the base of the urinary bladder. The central zone is continuous with the peripheral zone and, like the peripheral zone, is formed by several tubuloalveolar glands (mucosal, submucosal, main prostatic) which are located around the urethra. The acinar tissue consists of large, irregularly shaped spaces; the walls have intraluminal ridges or septa. The cells of the central zone differ significantly from those of the peripheral zone. They have more opaque, granular cytoplasm and less distinct cell membranes. Their cell length varies, they have an irregular luminal border, and they appear more crowded. Their nuclei, which are slightly larger than those of the peripheral zone and stain paler, are displaced to variable levels from the basement membrane. Carcinoma seldom arises in the central zone.

Transition Zone: Glands in the transition zone are formed from the junction of the proximal and distal urethral segments. This zone is less than 5% of the glandular prostate. The transition zone is composed of two minute glandular regions which are lateral to the preprostatic sphincter and directly related to the proximal urethral

segment. The periurethral region is related to this zone and to the junction of the proximal and distal urethral segments. Periurethral ducts, which are responsible for the genesis of benign prostatic hyperplasia, are present. In this zone one observes a minimal number of glands. The transition zone and other periurethral glands are the exclusive site of origin of benign prostatic hypertrophy. The area near or within the sphincter almost invariably produces the most numerous and largest nodules. Ten to twenty percent of carcinomas may develop in the transition zone.

Stroma: The anterior fibromuscular stroma is nonglandular. It constitutes ⅓ of the prostatic tissue within the prostatic capsule but is in continuity with the detrusor muscle of the neck of the urinary bladder. It is heavily fixed with the anterior surfaces of the three glandular zones, and represents the periurethral gland region. The fibromuscular stroma is composed of striated and smooth muscles, as well as elastin and collagen.

Capsules of the Prostate

There are three capsules of the prostate; two (the true and false) are anatomic the third is pathologic. The true capsule is a very thin covering surrounding the gland in toto. The false capsule (periprostatic fascia or prostatic sheath) is an extraperitoneal fascia (visceral layer of endopelvic fascia). This capsule is continuous with 4 fasciae; anteriorly, fascia of the bladder, puboprostatic ligament,

laterally, arcus tendineus of the fascia pelvis, Posterior: fascia of Denonvilliers and inferiorly, superior fascia of the urogenital diaphragm. Between the true and false capsules is a venous plexus, the prostatic or pudental venous plexus.

Part of the normal aging process is progressive prostatic growth due to benign prostatic hyperplasia (BPH), the peripheral part of the prostate becomes compressed against the surrounding endopelvic connective tissue, forming a surgical capsule (pathologic capsule). When enucleation of the prostate is performed, the plane between the compressed peripheral tissue and the adenomatous tissue permits removal of the adenoma, leaving behind the peripheral condensed prostatic tissue and the anatomic capsule.

Prostate Vascular Supply

Arterial Supply: The blood supply of the prostate is derived primarily from inferior vesical artery. A branch of this artery enters the prostate laterally at the prostatovesical junction. This artery divides into two branches, the peripheral and the central. The peripheral branch serves the majority of the prostatic parenchyma; the central branch supplies the urethra and the periurethral tissues. Other arteries contributing rami to the prostate are the internal pudental and middle rectal arteries. The middle rectal artery is considered to be poorly named, since most of its blood goes to the prostate gland.

Venous Drainage: There is a rich venous plexus (prostatic plexus) between the prostate gland and the prostatic sheath. It communicates with the internal iliac venous system and the presacral veins. The prostatic venous plexus receives the deep dorsal penile vein and the veins of the base of the bladder. The vesical and internal iliac veins receive most of the venous blood.

Lymphatic Drainage: From the prostatic acinus, large intraprostatic trunks are formed. These penetrate the prostatic capsule and form the periprostatic lymphatic plexus. This plexus yields lymphatic vessels which follow the vascular network of the prostatovesical arteries. The lymph vessels that follow the prostatovesical arteries travel to the internal iliac lymph nodes. The vessels also travel to the presacral lymph nodes and, occasionally, to the external iliac lymph nodes.

Prostate – Innervation

The preganglionic sympathetic nerve supply to the smooth muscle of the seminal vesicles, ejaculatory ducts, and prostate gland arises in the intermediate gray area of spinal cord levels L1 and L2 (or L3). Postganglionic fibres arise in the preaortic or pelvic plexuses. The sympathetic fibres cause contraction of the smooth muscle and expulsion of seminal fluid. Parasympathetic fibres from sacral cord levels S2, S3, and S4 synapse in pelvic ganglia and periprostatic ganglia.

They act perhaps to dilate blood vessels and stimulate secretion from glands of the genital system, including the prostate.

Histology of Prostate

Seventy percent of the weight of the prostatic mass is glandular epithelium. Thirty percent is fibromuscular, mainly non-striated. The glandular part contains ducts and acini which are lined with columnar epithelium and drain in the posterior and lateral walls of the prostatic urethra. In all regions, ducts and acini are lined with secretory epithelium, with a layer of basal cells and interspersed endocrine-paracrine cells beneath. The peripheral zone has small, rounded, uniform glands. The central and transitional zones have very large and irregular acini.

Physiology of Prostate

The prostate gland secretes a milk like alkaline fluid. This fluid is very important for the fertilization of the ovum, since sperm within both the ductus deferens and vaginal tissue produce fertilization-inhibiting acidity. Guyton stated that prostatic fluid most likely neutralizes the acidity of the fluids of the ductus deferens and vagina after ejaculation, enhancing the motility and fertility of the sperm. The prostatic fluid also contains citric acid, calcium, phosphorus, and other substances.

BENIGN PROSTATIC HYPERPLASIA [9]

Benign prostatic hyperplasia (BPH) is a pathologic process that contributes to, but is not the sole cause of, lower urinary tract symptoms (LUTS) in aging men. Despite intense research efforts in the past five decades to elucidate the under lying etiology of prostatic growth in older men, cause-and-effect relationships have not been established. For example, androgens are a necessary but not a clearly causative effect of BPH.

The nomenclature of voiding dysfunction in aging men is confusing and often inaccurate. The term BPH should be used with reference to the histologic process of hyperplasia, which can be demonstrated microscopically. Men with benign prostatic enlargement (BPE) presumably have an increase in total prostate volume because of BPH. BPE may or may not produce clinically significant LUTS and may or may not produce urodynamically proven bladder outlet obstruction.

ETIOLOGY OF BENIGN PROSTATIC HYPERPLASIA

BPH is but one cause of the LUTS in aging men commonly, and probably incorrectly, referred to as prostatism. Histopathologically, BPH is characterized by an increased number of epithelial and stromal cells in the periurethral area of the prostate. The observed increase in cell number may be due to epithelial and stromal proliferation or to impaired programmed cell death leading to cellular accumulation. Androgens, estrogens, stromal-epithelial interactions, growth

factors and neurotransmitters may play a role, either singly or in combination, in the etiology of the hyperplasic process.

Hyperplasia

In a given organ, the number of cells, and thus the volume of the organ, is dependent upon the equilibrium between cell proliferation and cell death. An organ can enlarge not only by an increase in cell proliferation but also by a decrease in cell death. The relative role of cell proliferation in human BPH is questioned because there is no clear evidence of an active proliferative process. Although it is possible that he early phases of BPH are associated with a rapid proliferation of cells, the established disease appears to be maintained in the presence of an equal or reduced rate of cell replication and increased expression of antiapoptotic pathway genes. Androgens not only are required for normal cell proliferation and differentiation in the prostate but also actively inhibit cell death. The hyperplasia results in a remodelling of the normal prostatic architecture. Epithelial budding from pre-existing ducts and the appearance of mesenchymal nodules characterize the early stages of the process. When the proliferating cell matures through a process of terminal differentiation, they have a finite life span before undergoing programmed cell death. In this paradigm, the aging process induces a block in this maturation process so that the progression to terminally differentiated cells is reduced, reducing the overall rate of cell death.

The Role of Androgens

Although androgens do not cause BPH, the development of BPH requires the presence of testicular androgens during prostate development, puberty, and aging patients castrated prior to puberty or who are affected by a variety of genetic disease that impair androgen action or production do not develop BPH. It is also known that prostatic levels of dihydrotestosterone (DHT) as well as the androgen receptor (AR) remain high with aging despite the fact that peripheral levels of testosterone are decreasing. Moreover, androgen withdrawal leads to partial involution of established BPH. Assuming normal ranges, there is no clear relationship between the concentration of circulating androgens and prostate size in aging men.

In the brain, skeletal muscle, and seminiferous epithelium, testosterone directly stimulates androgen-dependent processes. In the prostate, however, the nuclear membrane bound enzyme steroid 5 α –reductase converts the hormone testosterone into DHT, the principal androgen in this tissue. Ninety percent of total prostatic androgen is in the form of DHT, principally derived from testicular androgens. Inside the cell, both testosterone and DHT bind to the same high-affinity androgen receptor protein. The hormone receptor then binds to specific DNA binding sites in the nucleus, which results in increased transcription of androgen-dependent genes and ultimately stimulation of protein synthesis. Conversely androgen withdrawal from androgen-sensitive tissue results in a

decrease in protein synthesis and tissue involution. Besides inactivation of key androgen-dependent genes (e.g., prostate-specific antigen), androgen withdrawal leads to the activation of specific genes involved in programmed cell death. Despite the importance of androgens in normal prostatic development and secretory physiology, there is no evidence the either testosterone or DHT serves as the direct mitogen for growth of the prostate in older men. However, many growth factors and their receptors are regulated by androgens. Thus the action of testosterone and DHT in the prostate is mediated indirectly through autocrine and paracrine pathways.

Androgen Receptors

The prostate, unlike other androgen dependent organs, maintains its ability to respond to androgens throughout life. AR levels in the prostate remain high throughout aging. In fact, there is evidence to suggest that nuclear Androgen Receptor levels may be higher in hyperplastic tissue than in normal controls. Age-related increases in estrogen, as well as other factors, may increase AR expression in the aging prostate, leading to further growth.

5 α reductase enzyme

Two steroid 5 α reductase have been discovered, each encoded by a separate gene. Type 1 5 α-reductase, the predominant enzyme in extraprostatic tissues, such as skin and liver, Type 2 5α-rducatase is the predominant prostatic 5α-reductase. It is exquisitely sensitive to inhibition by finasteride and dutasteride. The type 2

enzyme is critical to normal development of the prostate and hyperplastic growth later in life.

Immunohistochemical studies with type 2 5α-reducatase specific antibodies show primarily stromal cell localization of the enzyme. Epithelial cells uniformly lack type 2 protein. The stromal cell plays a central role in androgen-dependent prostatic growth and that the type 2 5α-reductase enzyme within the stromal cell is the key androgenic amplification step. Thus, a paracrine model for androgen action in the gland is evident. In addition, it is possible that circulating DHT produced in the skin and liver may act on prostate epithelial cells in a true endocrine fashion.

Polymorphism in the type 2 5α-reductase enzyme (SRD5A2) has been reported, but their linkage to BPH is uncertain. Androgen withdrawal may partially exert its effect on the prostate through vascular effects. There is indirect evidence to suggest that abnormalities in the prostatic vascular system produced by other disease states may be a risk factor of BPH.

The Role of Estrogens

There is animal model evidence to suggest that estrogens play a role in the pathogenesis of BPH. The role of estrogens in the development of human BPH, however, is less clear.

Serum estrogen levels increase in men with age, absolutely or relative to testosterone levels. There is also suggestive evidence that intraprostatic levels of estrogen are increased in men with BPH. Patients with larger volumes of BPH tend

to have higher levels of estradiol in the peripheral circulation. At present, however, the role of estrogens in human BPH is not as firmly established as the role of androgens.

Stromal-Epithelial Interaction

There is abundant experimental evidence to demonstrate that prostatic stromal and epithelial cells maintain a sophisticated paracrine type of communication. This is strong evidence that one class of stromal cell excretory protein partially regulates epithelial cell differentiation. Thus, BPH may be due to a defect in a stromal component that normally inhibits cell proliferation, resulting in loss of a normal "braking" mechanism for proliferation.

The process of new gland formation in the hyperplastic prostate suggests a "reawakening" of embryonic processes in which the underlying prostatic stroma induces epithelial cell development. Many of the prostatic stromal-epithelial interactions observed during normal development and in BPH may be mediated by soluble growth factors or by the extracellular matrix (ECM). CRY61 (An Early Immediate Reponses Gene) is an ECM-associated protein that promotes adhesion, migration, and proliferation of epithelial and stromal cells. CRY61 expression is significantly increased in human BPH tissues and is induced by lysophosphatidic acid (and endogenous lipid growth factor).

Growth Factors:

Growth factors are small peptide molecules that stimulate, or in some cases inhibit, cell division and differentiation processes; Cells that respond to growth factors have on their surface receptors specific for that growth factor that in turn are linked to a variety of transmembrane and intracellular signalling mechanisms. Interaction between growth factors and steroid hormones may alter the balance of cell proliferation versus cell death to produce BPH.

In addition to FGF-2, acidic FGF (FGF-1), Int-2 (FGF-3), keratinocyte growth factor (KGF, FGE-7), transforming growth factors (EGF) have been implicated in prostate growth. Similar mechanisms may be operational in BPH, leading to the accumulation of epithelial cells. If cellular proliferation is a component of the BPH process, it appears that growth stimulator factors such as FGR-1, -2, -7, and -17 families; vascular endothelial growth factor (VEGF); and insulin-like growth factor (IGF) may play a role, with DHT augmenting or modulating the growth factor effects. In contrast, TGF-β, which is known to inhibit epithelial cell proliferation, may normally exert a restraining influence over epithelial proliferation that is lost or down regulated in BPH.

Although data on the absolute level of growth factor and growth factor receptors in hyperplastic as opposed to normal tissue are conflicting, it is likely that growth factors play some role in the pathogenesis of BPH.

Other Signalling Pathways

Sympathetic signalling pathways are important in the pathophysiology of LUTS. In addition, there is increasing evidence that sympathetic pathways may be important in the pathogenesis of the hyperplastic growth process. Alpha blockade, in some model systems, can induce apoptosis. Adrenergic pathways can also modulate the smooth muscle cell phenotype in the prostate all the components of the renin-angiotensin system (RAS) are present in prostatic tissue and may be activated in BPH. Either with or without sympathetic modulation, local RAS pathways may contribute to cell proliferation and smooth muscle contraction.

The Potential Role of Inflammatory Pathways and Cytokines in Benign Prostatic Hyperplasia

An additional source of growth factors in human BPH tissue may be the inflammatory cell infiltrates seen in many men with BPH. Thayer and associates (1992) reported extensive infiltration of human BPH tissues by activated T cells. Peripheral blood and tumor infiltrating T cells are known to express VEGF, a potent epithelial mitogen T cells are known to produce and secrete a variety of other growth factors including HB-EGF and bFGF/FGF-2. Thus T cell present in the local prostate environment were thought to be capable of secreting potent epithelial and stomal mitogens that promote stromal and glandular hyperplasia

A large number of cytokines and their receptors are seen in BPH tissue. Specifically, significant levels of IL-2, IL-4, IL-7, IL-17, interferon γ (IFEN- γ)

and their relevant receptors are found in BPH tissue. Macrophage inhibitory cytokine I is expressed in normal prostate tissue but significantly down regulated in BPH. To date, however, no firm cause-and-effect relationships have been established between prostatic inflammation and related cytokine pathways and stromal-epithelial hyperplasia.

Genetic and Familial Factors

There is substantial evidence the BPH has an inheritable genetic component. Sanda and colleagues (1994) conducted a retrospective case-control analysis of surgically treated BPH patients and control subjects at Johns Hopkins. The BPH patients were men whose respected prostate weights were in the highest quartile (greater than 37 g) and whose age at prostatectomy was in the lowest quartile. The hazard-function ratio for surgically treated BPH among first-degree male relatives of the controls was 4.2 (95% confidence interval [CI] demonstrating a very strong relationship [9, 10].

A segregation analysis showed that the results were most consistent with an Autosomal Dominant inheritance pattern. Approximately 50% of men undergoing prostatectomy of BPH at less the 60 years of age could be attributable to inheritable form of disease. In contrast, only about 9% of men undergoing prostatectomy of BPH at more than 60 years of age would be predicated to have a familial risk [9, 10]. However, the specific gene or genes involved in familial BPH or

that contribute to the risk of significant prostatic enlargement in sporadic disease remain to be elucidated.

Other Etiologic Factors

Androgens and soluble growth factors are clearly not the only important factors for the development of BPH. All mammalian prostates studied have testosterone, DHT, and AR as well as most of the known growth factor signalling pathways; however, only dog and man develop BPH. Interestingly, another glandular organ that remains androgen responsive throughout life, the seminal vesicle, does not develop hyperplasia. Obviously, other mechanisms or cofactors must be present in these two unique species making them susceptible to the disease. Non androgenic substances from the testis perhaps transmitted through the vas deferens or deferential blood vessels, for example, may play some role. Prolactin has long been speculated to play a role in BPH because of the known effects of this hormone on prostate cells in vitro. However, despite the documented presence of prolactin receptors in the human prostate and low circulating levels of the hormone, the role of prolactin in human prostate disease is unclear.

PATHOPHYSIOLOGY OF BENIGN PROSTATIC HYPERPLASIA[9]

The pathophysiology of BPH is complex. Prostatic hyperplasia increases urethral resistance, resulting in compensatory changes in bladder function. However, the elevated detrusor pressure required to maintain urinary flow in the

presence of increased outflow resistance occurs at the expense of normal bladder storage function. Obstruction induced changes in detrusor function, compounded by age- related changes in the bladder and nervous system function, lead to urinary frequency, urgency, and nocturia, the most bothersome BPH-related complaints.

Pathology[9]

Anatomic Features: BPH first develops in the periurethral transition zone of the prostate. The transition zone consists of two separate glands immediately external to the preprostatic sphincter and the main urethral wall at the point of urethral angulation near the verumontanum.

All BPH nodules develop either in the transition zone or in the periurethral region. Although early transition zone nodules appear to occur either as the disease progresses and the number of small nodules increases, they can be found in almost any portion of the transition or periurethral zone. However, the transition zone also enlarges with age unrelated to the development of nodules.

One of the unique features of the human prostate is the presence of the prostatic capsule, which plays an important role in the development of LUTS, In the dog, the only other species known to develop naturally occurring BPH, symptoms of bladder outlet obstruction and urinary symptoms rarely develop because the canine prostate lacks a capsule. Presumably the capsule transmits the "Pressure" of tissue expansion to the urethra and leads to an increase in urethral resistance. Thus, the clinical symptoms of BPH in man may be due not only to

age-related increases intraprostatic size but also to the unique anatomic structure of the human gland. Clinical evidence of the importance of the capsule can be found in series that clearly document that incision of the prostatic capsule (transurethral incision of the prostate) results in a significant improvement in outlaw obstruction, despite the fact that the volume of the prostate remains the same.

Histologic Features: BPH is a true hyperplastic process. Histologic studies document and increase in the cell number the majority of early periurethral nodules are purely stomal in character. It is unclear whether these early stromal nodules contain mainly fibroblast-like cells or whether differentiation toward a smooth muscle cell type is occurring. In contrast, the earliest transition zone nodules represent proliferation of glandular tissue that may be associated with an actual reduction in the relative amount of stroma.

These glandular nodules are apparently derived from newly formed small duct branches that bud off from existing ducts, leading to a totally new ductal system within the nodule. This type of new gland formation is quite rare outside embryonic development. This proliferative process leads to a tight packing of glands within a given area as well as an increase in the height of the lining epithelium.

During the first 20 years of BPH development, the disease may be predominantly characterized by an increased number of nodules, and the subsequent growth of each new nodule is generally slow. Then a second phase of

evolution occurs in which there is a significant increase in the size of nodules. In the first phase, the glandular nodules tend to be larger than the stromal nodules. In the second phase, when the size of individual nodules is increasing, the size of glandular nodules clearly predominates.

The Bladder's Response to obstruction

Current evidence suggests that the bladder's response to obstruction is largely an adaptive one. However, it is also clear that many lower tract symptoms in men with BPH or prostate enlargement are related to obstruction – induced changes in bladder function rather than to outflow obstruction directly.

Obstruction-induced changes in the bladder are of two basic types. First, the changes that lead to detrusor instability or decreased compliance and second, the changes associated with decreased detrusor contractility, which are associated with further deterioration in the force of the unitary stream, hesitancy, intermittency, increased residual urine, and (in a minority of cases) detrusor failure. Acute urinary retention should not be viewed as inevitable result of this process. Many patients presenting with acute urinary retention have more than adequate detrusor function, with evidence of a precipitating event leading to the obstruction.

There is considerable evidence that the response of the detrusor smooth muscle cell to stress (increased load related to outlet obstruction) is not as adaptive as the response of skeletal muscle to stress. In the latter case, a relatively normal repertoire of contractile protein genes are upregulated and an increased number of

normally organized contractile units assemble in the muscle cell. In the detrusor smooth muscle cell load induced hypertrophy leads to change in myosin heavy chain isoform expression and to a signification alteration in the expression of a variety of thin filament-associated proteins.

In addition to obstruction – induced changes in the smooth muscle cell and ECM of the bladder, there is increasing evidence that obstruction may modulate neural – detrusor responses as well

EPIDEMILOGY AND NATURAL HISTORY

Definitions

The study of epidemiology is concerned with the distribution and determinants of diseases in humans. Form this evolve the components for descriptive epidemiology, description of disease incidence, mortality, and prevalence by person, place, and time, and analytic epidemiology, the search for determinants of disease risk that may sever to increase prospects for prevention.

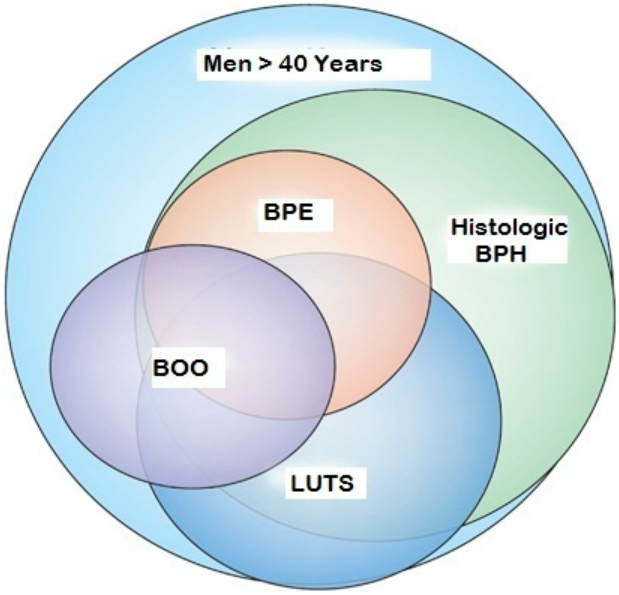
There is no globally accepted epidemiologic definition of BPH, and thus prevalence and incidence rates must be viewed in the context of the definitions chosen by the investigator reporting the data.

Cross-Sectional Studies of Clinical Prevalence:

Descriptive epidemiology relies on the presence of a single universally accepted definition of “disease.” The definitions of BPH, however, have undergone several changes in the past decade, and, at present, no single criterion can be

applied. In the past, the term “prostatism” was used, incorrectly referring to the prostate as the sole source of the typical LUTS founds in aging men. It has been pointed out that there are at least three interrelated phenomena that can be assessed independently, namely the symptoms (formerly called prostatism), enlargement of the prostate gland, and presence of obstruction. In a given patient, all three, two of the three, or only one of the three entities might be present. Paul Abrams coined the term lower urinary tract symptoms to replace the old and inappropriate term prostatism. The same patients then can be further classified based on the degree of prostatic enlargement as measured by digital rectal examination (DRE), transrectal ultrasonography (TRUS), or magnetic resonance imaging (MRI) and lastly by the presence and degree of bladder outlet obstruction as measured by flow rate recordings or invasive pressure flow studies.

The diagram [9, 11] shown below attempts to illustrate the difficulties in using different disease definitions. Of all men older than 40, a certain proportion develop histology hyperplasia of the prostate, that is BPH. Of those, some but not all develop LUTS, and other may have LUTS for reasons other than BPH**.** Prostate enlargement occurs in some but again not all men with histologic BPH and LUTS, and some men with enlarged glands may not have any symptoms at all. Lastly, urodynamically proven obstruction may be present in men who have either one several or all of histology BPH, LUTS, and enlarged glands, yet other may have obstruction without having any evidence of BPH.



In addition to the mere enumeration of symptoms by frequency of occurrence, the bother associated with the symptoms, interference with activities of daily living, and the impact the symptoms have on quality of life are important distinguishing characteristics.

Accordingly, when studying the prevalence of clinical BPH admittedly an imprecise tem describing the constellation of LUTS, bother, interference, quality of life impact, with or without enlargement, obstruction, and so forth disease definitions may be applied that take either one or several of these items into consideration. For the subsequent discussion it is important to recognize that very few if any clear cut-off points have been established that allow differentiation between disease absent or present states (e.g., one might argue that a prostate volume over 30mlconstitutes clinical BPH, but others might argue for a higher or

lower cut-off point; similar observations apply for symptoms and degrees of obstruction).

Symptom Severity and Frequency[9, 17]

Form a pragmatic point of view, studies of symptom severity and frequency are of greatest importance in a disease that is rarely fatal and is characterized by its effects on the quality of life. The development, validation, and translation with cultural and Linguistic validation of the standardized, self-administered seven item American Urological Association (AUA) symptom index (also known as the International Prostate Symptom Score [IPSS]) has been a pivotal event in the clinical research on LUTS and BPH.

With the total score running from 0 to 35 pints, patients scoring 0 to 7 points are classified as mildly symptomatic, those scoring from 8 to 19 points as moderately symptomatic, and those scoring 20 to 35 points as severely symptomatic. The instrument is an integral part of virtually every epidemiologic study as well as treatment studies in the field, and the availability of validated translations in many common languages allows cross-cultural comparisons of unprecedented scope. Socioeconomic factors do not seem to influence responses to the questionnaire and fundamentally similar responses are obtained when the questionnaire is self-administered, read to the patient mailed in, or administered in some other way.

In the past month:	Not at All	Less than 1 in 5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
Incomplete Emptying: How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
Frequency: How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
Intermittency: How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency: How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
Weak Stream: How often have you had a weak rinary stream?	0	1	2	3	4	5	
Straining: How often have you had to strain to start urination?	0	1	2	3	4	5	
	Nil	1 **Time**	2 **Time**	3 **Time**	4 **Time**	5 **Time**	
Nocturia: How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total IPSS Score							

Score: 1-7: Mild 8-19: Moderate 20-35: Severe

Apart from these 7 questions regarding symptom severity International Prostate Symptom Score also contains an 8th question which is a Quality Of Life (QOL) question. This question is added as the International Scientific Committee (SCI), under the patronage of the World Health Organization (WHO) and the International Union Against Cancer (UICC), recommends the use of only a single question to assess the quality of life. The answers to this question range from "delighted" to "terrible" or 0 to 6.

Quality of Life Due to Urinary Symptoms	Delighted	Pleased	Mostly Satisfied	Mixed	Mostly Dissatisfied	Unhappy	Terrible
If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that?	1	2	3	4	5	6	

A very large international investigation of LUTS in Asian men was undertaken by Homma and colleagues (1997) in which 7588 men from Japan, China, Taiwan, Korea, the Philippines, Thailand, Singapore, Pakistan, India, and Australia were, queried. The finding of 18%, 29% , 40% and 56% of men in their 40s, 50s, 60s, and 70s having moderate to severe symptoms is line with the other studies reported both form Asia and from Europe and North America.

Prostate Size

Prostate size can be estimated by DRE, although the reliability across observers is in general considered poor. In addition, DRE tends to underestimate true prostate size as determined by TRUS or other imaging modalities. The magnitude of the underestimation increases with increasing prostate size for 25% up to 50% or more For the purpose of epidemiologic studies, TRUS and MRI measurements are preferred, although MRI measurement are somewhat expensive when attempting cross-sectional examinations of populations. TRUS volume measurements using the prostate ellipsoid volume formula are the most widely accepted measure of prostate volume with reasonable statistical performance characteristics, particularly when performed by a single or several well trained examiners.

Measures of obstruction

Subvesical obstruction can be measured only by invasive pressure-flow studies; nonintubated free flow rates provide at best an indirect measure for the probability of obstruction being present. Unfortunately, no large-scale cross-sectional studies have been done employing pressure-flow tests because of the invasive and costly nature of the test, and it is unlikely that significant data sets will ever become available.

It is commonly accepted that a maximum flow rate of less than 10ml/sec indicates a high probability of obstruction and a flow rate of greater than 15ml/sec indicate a low probability, with 10 to 15 ml/sec presenting an intermediate range.

Post Residual Urine Volume[19, 20]

One of the important subjects of tests for urinary incontinence is the post void residual urine volume (PVR), the amount of urine left after urination. Normally, about 50 mL or less of urine is left; more than 200 mL is a definite sign of abnormalities. Measurements in between require further tests. The most common method for measuring PVR is with a catheter, a soft tube, which is inserted into the urethra within a few minutes of urination. PVR can also be measured using transabdominal ultrasonography.

A study on the distribution of Post – void residual urine volume in randomly selected men published in the Journal Of Urology suggests little variation in the distribution of post-void residual urine volume across age groups or levels of urinary symptoms and peak urinary flow rate. However, a somewhat stronger relationship was found between residual urine and prostate volume.

Prostate-specific antigen (PSA) [8, 12, 18]

Prostate-specific antigen (PSA) is a protein produced by normal prostate cells. This enzyme participates in the dissolution of the seminal fluid coagulum and

plays an important role in fertility. The highest amounts of PSA are found in the seminal fluid; some PSA escapes the prostate and can be found in the serum.

The level of PSA in the blood can be determined by a simple blood test. PSA blood test results are reported as nanograms per millilitre or ng/ml. Normal level usually range from 0 ng/ml to 4 ng/ml.

Rising levels of PSA in serum are associated with prostate cancer. The PSA level also tends to rise in men with benign prostatic hyperplasia (BPH) and is a good marker for prostate volume. PSA levels are usually elevated in men with acute bacterial prostatitis.

As BPH is a true hyperplasia, more cells produce a greater amount of PSA. It has been suggested that many PSA elevations detected and investigated in clinical practice may in fact be due to BPH leading to the argument that PSA is a better marker of BPH than of prostate cancer. One approach to distinguish the two conditions when PSA is elevated is to perform a free-to-total PSA ratio: more free PSA than complexed PSA suggests BPH rather than prostate cancer. A ratio of around 20% or greater for free PSA is considered more likely to represent BPH than cancer. PSA is discussed further in the investigations section below.

Correlations between Parameters

As noted, all relevant parameters such as symptom severity and frequency, bother, interference, disease-specific health-related quality of life (HROQOL),

maximum flow rate, and prostate volume tend to worsen with advancing age. However, reported correlations between these parameters as well as urodynamic pressure-flow studies are in general weak with some exceptions. Strong correlations exist between measures of symptom severity and frequency (I-PSS score), bother, disease-specific HRQOL, and interference scores.

With the exception of age, correlations between various measures of LUTS and BPH are modest in community based population studies and weak in BPH clinic and trial populations, not precluding, however, a clinical meaningful relationship. The relationship between serum PSA and prostate volume is moderate and dependent on age and racial and ethnic origin. Neither symptoms nor flow rate nor prostate volume measures can predict presence and degree of obstruction reliably.

MATERIALS AND METHODS

150 male patients aged more than 50 years admitted with inguinal hernia to the 6th Surgical Unit of Government Rajaji Hospital, Madurai between September 2009 and September 2011 are selected as cases.

Inclusion criteria were,

1. Male sex,
2. Age more than 50 years,
3. Those with inguinal hernia.

Exclusion criteria were,

1. Female sex,
2. Age ≤ 50 years,
3. Known case of connective tissue disorders,
4. Known case of BPH, who are already on drugs or have had any form of surgery for BPH in the past.
5. Presence of complications of hernia, such as irreducibility, strangulation or obstruction.

The method of selection was to select the first 150 male patients in the order date of their admission to the Government Rajaji Hospital without any other

methods of randomization. The case selection was independent of the side of the hernia or whether the hernia is unilateral, bilateral or recurrent.

Every week, after selecting cases, the corresponding number of control subjects was selected randomly from the patients admitted to the 6th Surgical Unit of Government Rajaji Hospital, Madurai for conditions other than inguinal hernias so as to make a control group of 150 subjects. In case, required number of controls was not available from the corresponding week's admission, the required number of controls was selected from the previous week's admission.

The inclusion criteria for the controls include,

1. Male sex,
2. Age > 50 years,
3. Not seriously ill.

The exclusion criteria for the controls include,

1. Female sex
2. Age ≤ 50 years,
3. Known case of connective tissue disorders,
4. Known case of BPH, who are already on drugs or have had any form of surgery for BPH in the past,
5. Presence of inguinal hernia unilateral, bilateral or recurrent,

6. History of surgery done for inguinal hernia in the past,
7. Seriously ill or bedridden patient.

Informed written consent was obtained from each of the cases and controls. All subjects were interviewed and examined by the single observer.

Very few if any clear cut off points have been established that allow differentiation between disease absent and present states [9]. Hence for this study three independent variable - International Prostate Symptom Score, Prostate-specific antigen (PSA), and prostate volume were taken and prevalence of BPH in cases and controls were found out for each of the three variables separately.

International Prostate Symptom Score:

International Prostate Symptom Score was obtained by reading out the questionnaire and answer options in the prescribed format as many of the study subjects were not able to read the questionnaire and mark the answers in the prescribed format. Each question was read out with its answer options and score for each question was marked separately and the sum is calculated to find out the International Prostate Symptom Score of each subject. The International Prostate Symptom Score (IPSS) is an 8 question (7 symptom questions + 1 quality of life question) written screening tool used to screen for, rapidly diagnose, track the symptoms of, and suggest management of the symptoms of the disease benign

prostatic hyperplasia (BPH). The score of 7 symptom questions were added to get the International Prostate Symptom Score (IPSS).

IPSS result of 7 symptoms questions

Score	Correlation
0 – 7	Mildly symptomatic
8 – 19	Moderately symptomatic
20 – 35	Severely symptomatic

Subjects who are moderately or severely symptomatic i.e., those with International Prostate Symptom Score ≥8 were taken as having significant benign prostatic hyperplasia. Those who are mildly symptomatic i.e. those with International Prostate Symptom Score <8 were taken as having no significant benign prostatic hyperplasia.

PROSTATE SPECIFIC ANTIGEN[8].

Prostate-specific antigen (PSA) is an enzyme produced by the prostate gland. Normally, PSA is secreted in small amounts into the bloodstream. However, larger amounts of PSA are released when the prostate gland is enlarged, infected,

or diseased. The level of PSA in the blood can be determined by a simple blood test. PSA blood test results are reported as nanograms per millilitre or ng/ml. Normal level usually range from 0 ng/ml to 4 ng/ml.

The blood samples from the subjects were taken before doing Digital Rectal Examination. Subjects with Prostate-specific antigen values > 4ng/ml were taken as having significant benign prostatic hyperplasia in this study. Those subjects whose Prostate-specific antigen values ≤ 4ng/ml were taken as having no significant benign prostatic hyperplasia.

PROSTATE VOLUME:

The normal weight of the prostate in a young adult is from 17 to 19g[4]. Since $1cm^3$ of prostate tissue equals approximately 1g of prostate tissue, >20cc of prostate volume was taken as significant benign prostatic hyperplasia in this study[9]. Since Trans Rectal Ultra sonogram is not available in our hospital trans abdominal ultra sonogram was used to measure the Prostate size in this study So subjects with prostate volume > 20cc are taken as having significant benign prostatic hyperplasia and those with prostate size ≤ 20cc are taken as having no significant benign prostatic hyperplasia.

International Prostate Symptom Score (IPSS), Serum Prostate-specific antigen (PSA), and Prostate volume were found out for all cases and controls.

The prevalence of benign prostatic hyperplasia in both cases and controls were done for each of the three variables studied here. Univariate analysis of the association between inguinal hernia and benign prostatic hyperplasia was done for each of the three variables separately.

Chi Square test is used to find out the association and P values were calculated for each variable. A P value <0.05 is taken as statistically significant.

ANALYSIS AND DISCUSSION

The study was conducted over a period of 2years from 1st August, 2009 to 31st August, 2011. A total no of 150 cases and 150 controls were selected. The cases were all males, aged between 51years and 85years with a mean age of 62.42years.

The age distribution of cases was as follows.

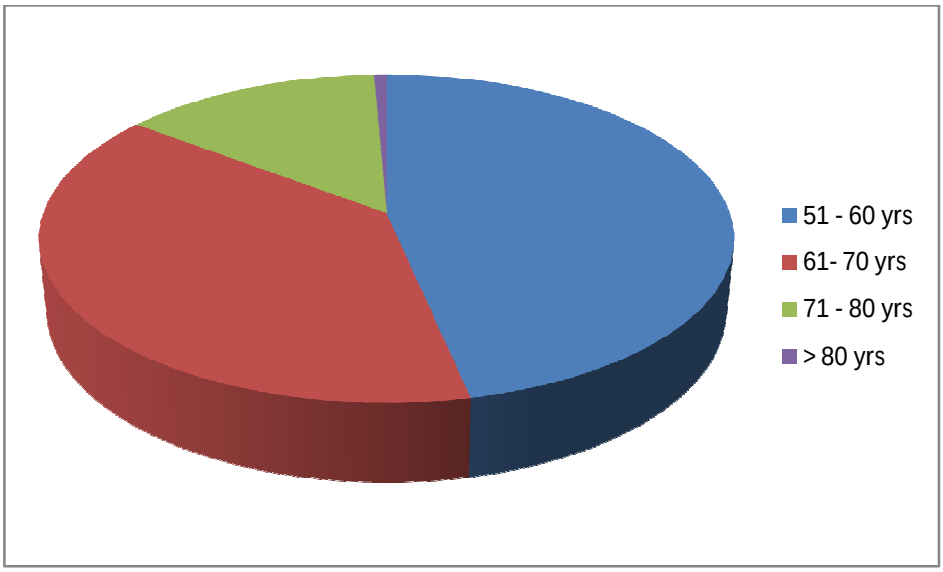
Age group	51 – 60 years	61- 70 yrs	71 - 80 yrs	> 80 yrs
Number of cases	70	58	21	1

The controls were all male subjects, aged between 51years and 83 years with a mean age of 63.56.

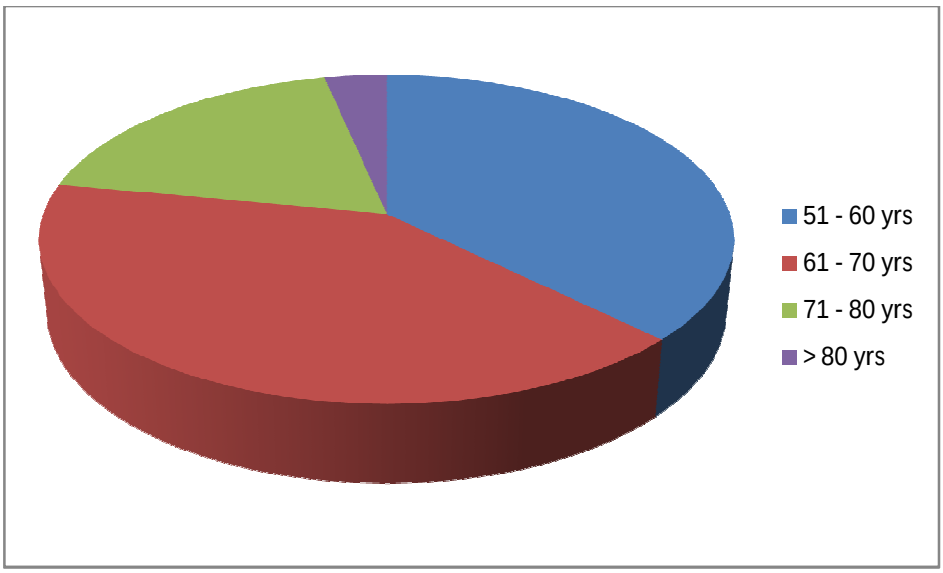
The age distribution of controls was as follows.

Age group	51 – 60 years	61- 70 yrs	71 - 80 yrs	> 80 yrs
Number of cases	56	61	28	5

The age distribution of cases

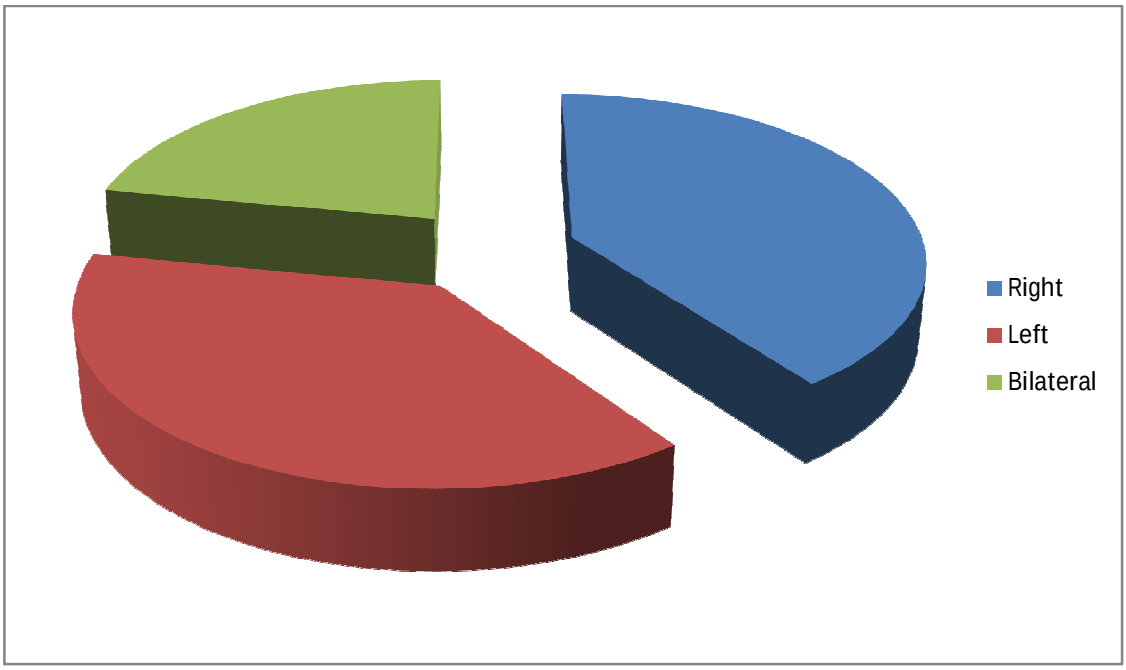


The age distribution of controls



The side of the hernia among cases were as follows

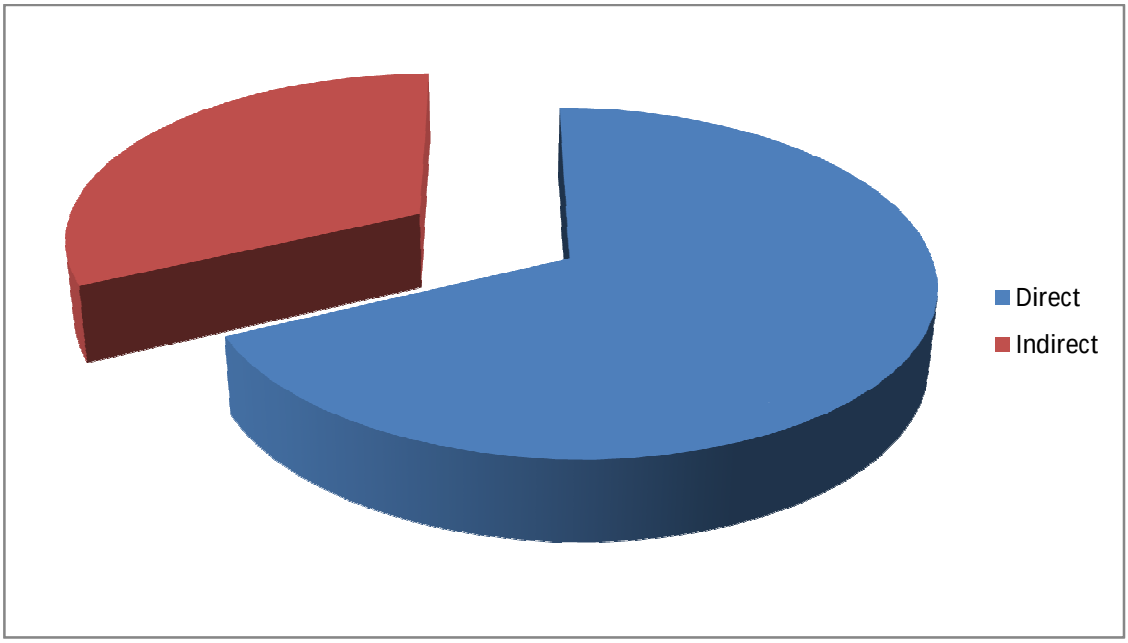
Side	Right	Left	Bilateral
Number of Subjects	60	57	33



There were 7 cases of recurrent hernias among the cases. Out of which 2 were bilateral, 4 left sided and 1 was right sided hernia. The remaining 143 hernias were new onset hernias.

Type of hernia – Direct or Indirect

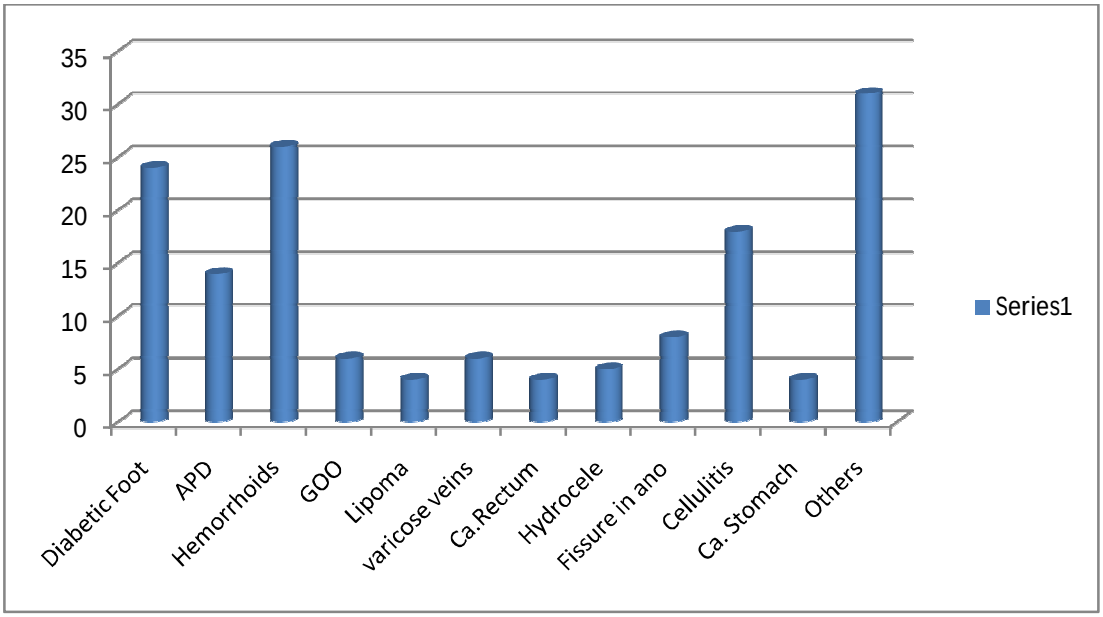
Type of Hernia	Direct	Indirect
Number of Subjects	102	48



All recurrent hernias were of direct variety only. All the subjects were having reducible hernia only.

The control subjects were also selected from the ward from those patients admitted for diseases other than Hernia. The control subjects were having different illness such as Diabetic Foot, Haemorrhoids, Hydrocele, APD, Acute Gastritis, Ca. Rectum, Chronic Pancreatitis, Fissure in ano, Ileocaecal TB, Gastric Outlet Obstruction, Cellulites, Varicose Veins, Liver Abscess, Ca. Stomach, POVD, Fournier's Gangrene, Lipoma, Malignant Melanoma, Basal Cell Carcinoma, Ca.Colon, Fistula in Ano, Obstructive Jaundice and Ca.Penis.

Diagnoses among controls

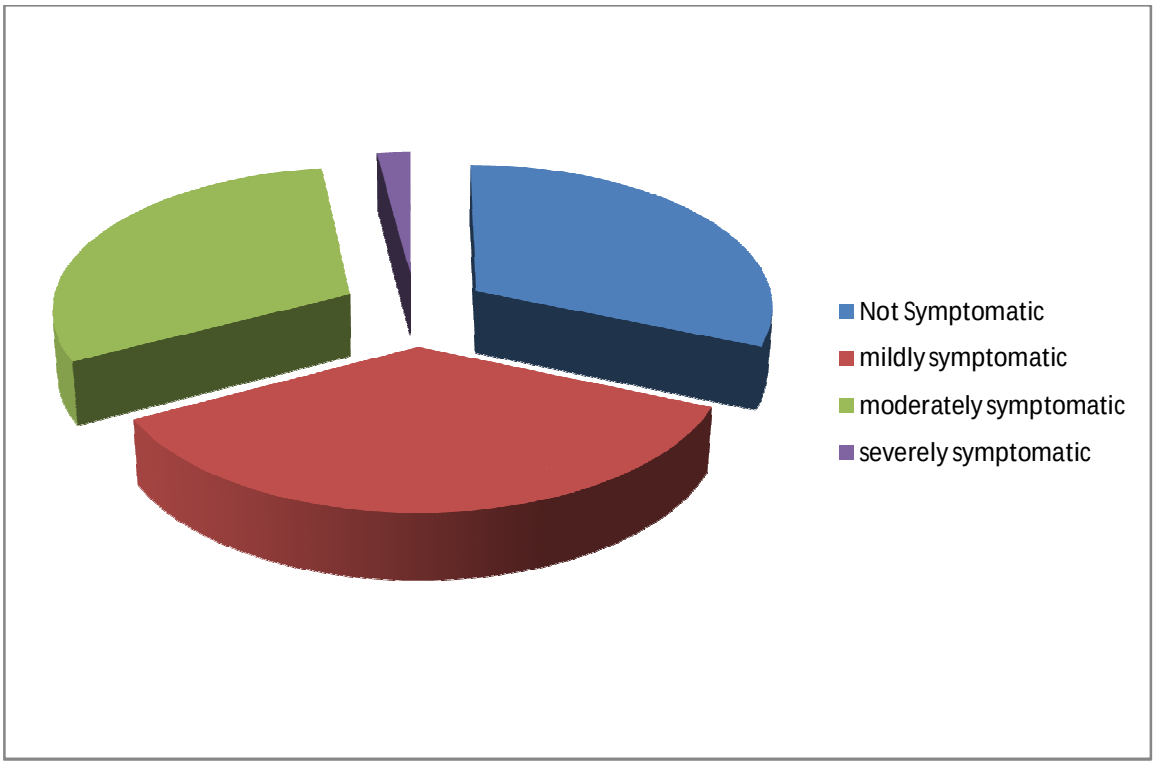


None of the control subjects had inguinal hernia or have had any kind of surgery for inguinal hernia in the past.

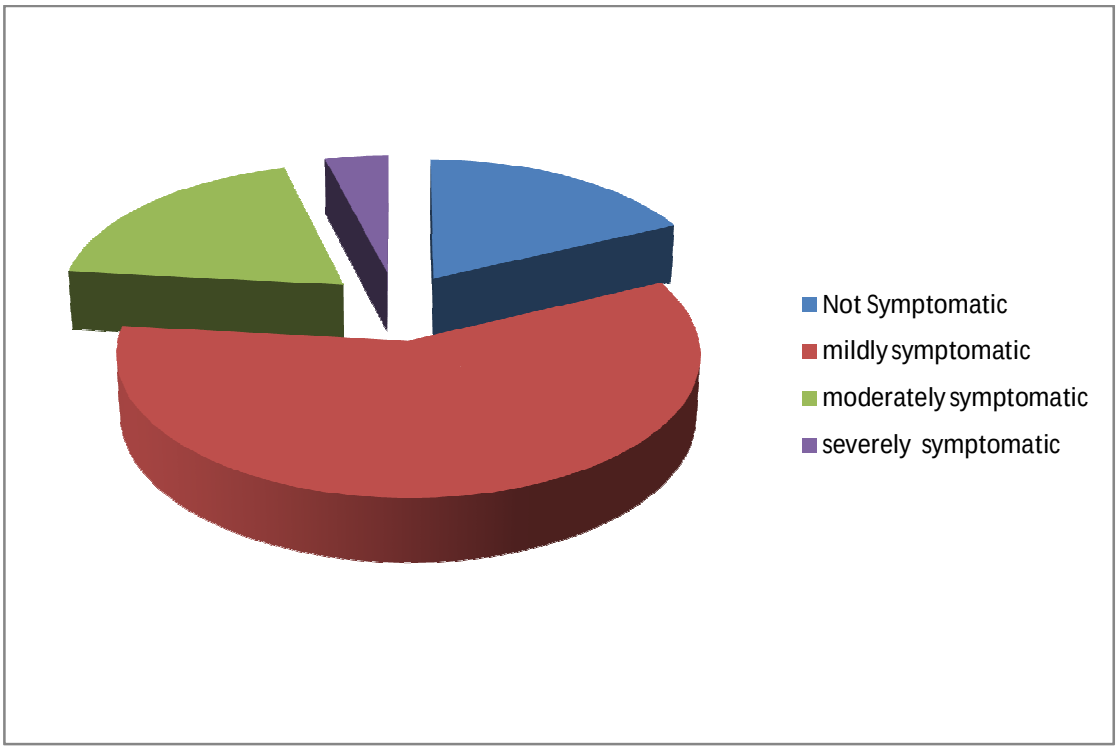
INTERNATIONAL PROSTATE SYMPTOM SCORE:

Among the cases 47 subjects were not symptomatic, 54 were mildly symptomatic, 46 were moderately symptomatic and 3 were severely symptomatic at the time of admission. Among controls, 115 were mildly symptomatic or having no symptoms, 29 were moderately symptomatic and 6 were severely symptomatic.

International Prostate Symptom Score of Cases:



International Prostate Symptom Score of controls



All subjects who were moderately or severely symptomatic were considered as having significant BPH. Hence there were 49 subjects among cases and 35 subjects among controls were having significant BPH.

Crosstab

			VAR00001		
			cases	controls	Total
IPSS	≥8	Count	49	35	84
		% within IPSS	58.3%	41.7%	100.0%
	<8	Count	101	115	216
		% within IPSS	46.8%	53.2%	100.0%
Total		Count	150	150	300
		% within IPSS	50.0%	50.0%	100.0%

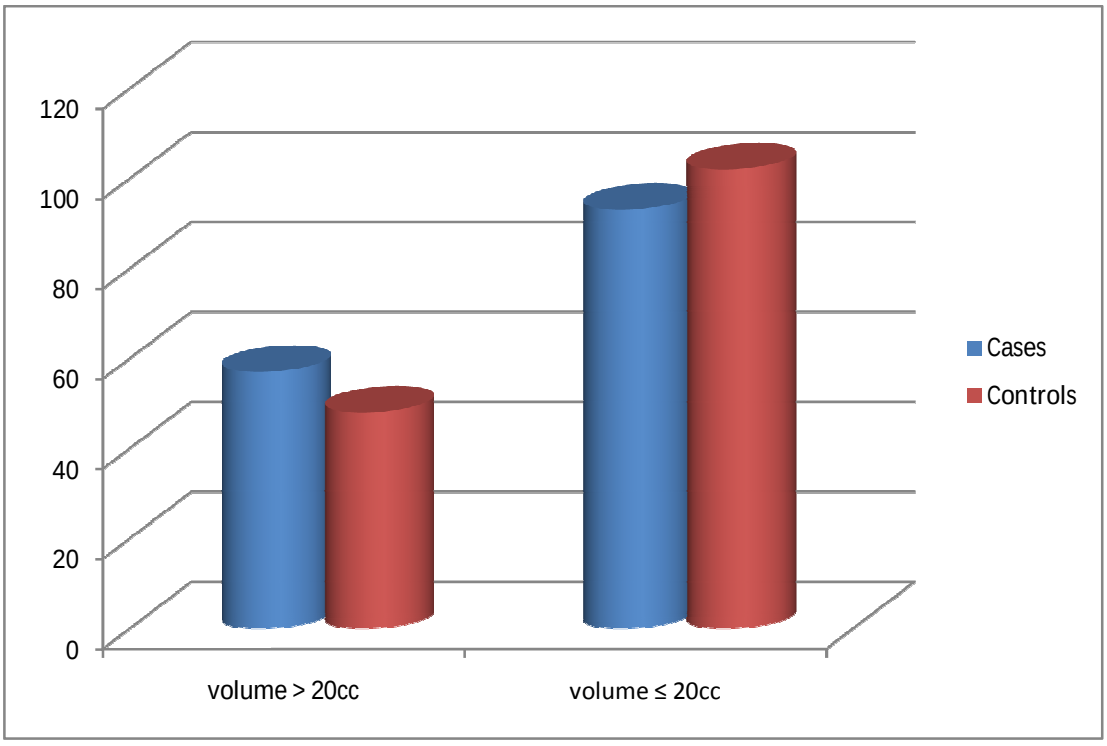
Chi square value= 3.24; P value 0.072

Among those scored ≥ 8 in IPSS scores 58.3% were cases and 41.75% were controls. However the difference was not found to be statistically significant.

PROSTATE VOLUME:

Among cases, 57 subjects were having prostate volume >20cc and 93 were having prostate volume ≤ 20cc. Therefore 57 of 150 cases were taken as having significant Benign Prostatic Enlargement.

Among controls, 48 subjects were having prostate volume > 20cc and 102 were having prostate volume ≤ 20cc. Therefore 48 of 150 controls were taken as having Benign Prostatic Enlargement.



Crosstab

			VAR00001		
			cases	controls	Total
Prostate volume	>20cc	Count	57	48	105
		% within volume	54.3%	45.7%	100.0%
	≤20cc	Count	93	102	195
		% within volume	47.7%	52.3%	100.0%
Total		Count	150	150	300
		% within volume	50.0%	50.0%	100.0%

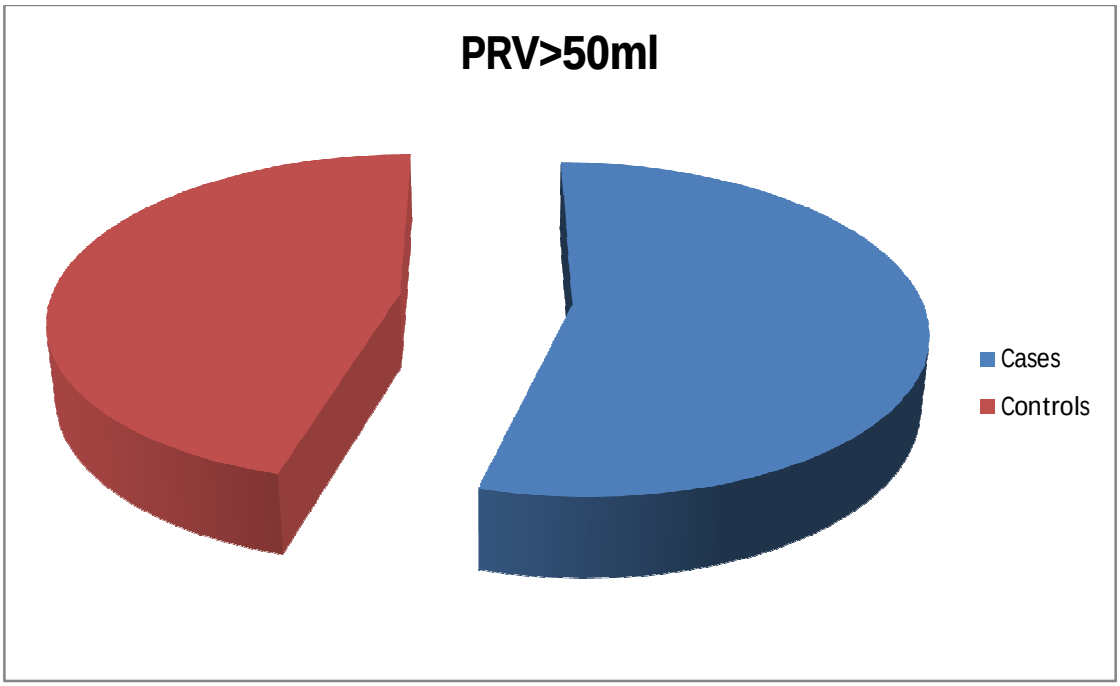
Chi square= 1.18; P value 0.276

Among those who had a prostate vol >20 ng/ml, 54.3% were cases and 45.7% were controls. However the difference is not statistically significant.

Post - Void Residual Urine Volume

The measurement of Post – void residual urine volume is done by Ultra sonogram of abdomen. A volume of > 50ml is taken as significant. But there was no significant difference in the distribution of the number of patients with Post – void residual urine volume >50ml between cases and controls (19 and 16 respectively).

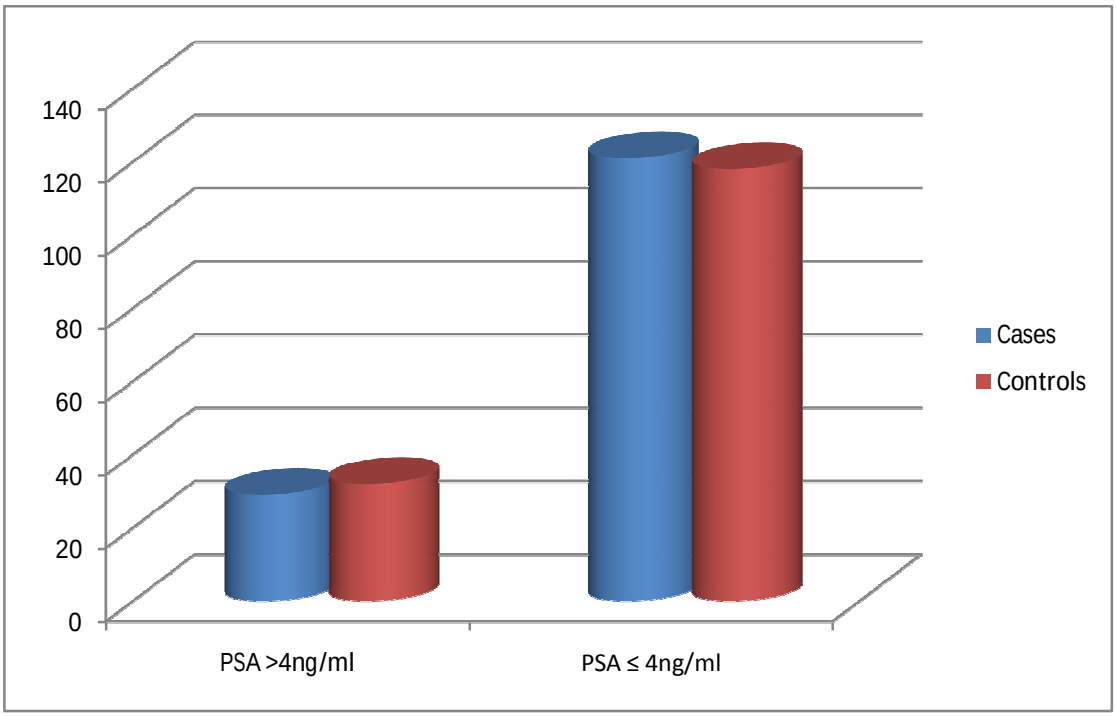
Distribution of Post - Void Residual Urine Volume among Cases and Controls:



PROSTATE SPECIFIC ANTIGEN:

Among the cases 29 subjects were having PSA > 4ng/ml and 121 were having PSA ≤ 4ng/ml. Therefore 29 out of 150 cases were taken as having significant Benign Prostatic Enlargement.

Among the controls 32 subjects were having PSA > 4ng/ml and 118 were having PSA ≤ 4ng/ml. Therefore 32 out of 150 controls were taken as having significant Benign Prostatic Enlargement.



Crosstab

			VAR00001		Total
			cases	controls	
PSA	> 4 ng/ml	Count	29	32	61
		% within PSA	47.5%	52.5%	100.0%
	≤4 ng/ml	Count	121	118	239
		% within PSA	50.6%	49.4%	100.0%
Total		Count	150	150	300
		% within PSA	50.0%	50.0%	100.0%

Chi square= 0.185; P value 0.665

Among those who had PSA value > 4ng/ml, 47.5% were cases and 52.5% were controls. However the difference is not statistically significant.

CONCLUSION

- Among the cases 117 were having unilateral hernia and 33 were having bilateral hernias.
- Right sided hernia was slightly more common than the left sided hernias (60 vs 57).
- Univariate analysis of association between Inguinal Hernia and Benign Prostatic Hyperplasia using International Prostate Symptom Score showed no statistically significant association between the two.
- Univariate analysis of association between Inguinal Hernia and Benign Prostatic Hyperplasia using Serum Prostatic Specific Antigen also showed no statistically significant association between the two.
- Prostatic size also did not show any statistically significant association between Inguinal Hernia and Benign Prostatic Hyperplasia.
- There was no significant difference in the distribution of the number of patients with Post – void residual urine volume >50ml between cases and controls.

- Although both Inguinal Hernia and Benign Prostatic Hyperplasia are seen with increased frequency in the aged male population, this study showed no statistically significant association between the two. Their occurrence together is considered a chance co-existence rather than cause and effect.

LIMITATIONS OF THE STUDY

- As the Uroflowmetric studies are not available in our hospital, it could not be used in this study. Univariate analysis using uroflowmetric analysis may better reflect the cause – effect association between Inguinal Hernia and Benign Prostatic Hyperplasia as it will better assess the obstructive nature of BPH.
- Age standardization could not be done in this study because of the method of selection of cases and controls.
- Trans Rectal Ultrasonographic assessment of prostatic size could not be done in this study as it was not available in our hospital.

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PROFORMA – CASES

Basic Details

Name: Age/Sex

IP NO: DOA:

Hernia

Right ◯ Left ◯ Bilateral ◯ Recurrent ◯

Direct ◯ Indirect ◯

IPSS:

In the past month:	Not at All	Less than 1 in 5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
Incomplete Emptying: How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
Frequency: How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
Intermittency: How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency: How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
Weak Stream: How often have you had a weak rinary stream?	0	1	2	3	4	5	
Straining: How often have you had to strain to start urination?	0	1	2	3	4	5	

	Nil	1 **Time**	2 **Time**	3 **Time**	4 **Time**	5 **Time**	
Nocturia: How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total IPSS Score							

Prostate Volume :

Post - void Residual Urine Volume :

PSA :

PROFORMA CONTROLS

Basic Details

Name: Age/Sex

IP NO: DOA:

Diagnosis:

IPSS:

In the past month:	Not at All	Less than 1 in 5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
Incomplete Emptying: How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
Frequency: How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
Intermittency: How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
Urgency: How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
Weak Stream: How often have you had a weak rinary stream?	0	1	2	3	4	5	
Straining: How often have you had to strain to start urination?	0	1	2	3	4	5	
	Nil	1 **Time**	2 **Time**	3 **Time**	4 **Time**	5 **Time**	

Nocturia: How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total IPSS Score							

Prostate Volume :

Post - void Residual Urine Volume :

PSA :

KEY TO THE MASTER CHART

A	:	Age
IP No	:	Inpatient number
Rt	:	Right sided
Lt	:	Left sided
B/L	:	Bilateral
Rc.	:	Recurrent Hernia
D	:	Direct Hernia
ID	:	Indirect Hernia
Ic.Em	:	Incomplete Emptying
Fr.	:	Frequency of Urination
In.Em	:	Intermittent Emptying
Urg.	:	Urgency
Wk.St.	:	Weak Stream
Str.	:	Straining
Noct.	:	Nocturia
IPSS	:	International Prostate Symptom Total Score
Pr.V	:	Prostate volume in cc
PVR	:	Post – void residual urine volume >50ml
PSA	:	Prostate Specific Antigen
n/ml	:	nanograms/litre

MASTER CHART CASES

S.No.	Name	A	IP NO	Hernia						International Prostate Symptom Score								Pr.V	PVR	PSA
				Rt	Lt	B/L	Rc.	D	ID	Ic.Em	Fr.	In.Em	Urg.	Wk.St.	Str.	Noct.	IPSS			
1	Chinnan	65	77278		*				*	0	1	0	1	1	1	0	4	18		0.3
2	Murugan	63	77268		*			*		0	0	0	0	1	1	1	3	22		1.6
3	Veeranan	70	79526	*					*	1	3	1	4	2	3	1	15	28		6.1
4	Gurusamy	57	79616	*					*	0	1	0	0	1	0	0	2	17		0.6
5	Andimuthu	64	79586	*				*		1	2	0	2	2	2	1	10	32	*	5.2
6	Baluchamy	65	79180	*					*	0	0	0	1	1	0	0	2	16		1.2
7	Shanmugam	75	79432	*				*		3	4	2	4	5	4	3	25	33	*	3.6
8	Jafer Khan	56	83816		*			*		0	0	0	0	0	0	0	0	17		1.1
9	Athigavalli	57	84803		*				*	1	1	1	2	2	3	1	11	24		3.9
10	Saravanamuthu	60	86809	*				*		0	1	0	1	0	0	0	2	17		4.2
11	Subburam	60	88444	*					*	0	1	0	0	1	0	0	2	22		0.6
12	Murugan	55	90736			*			*	0	0	0	0	0	0	0	0	24	*	0.9
13	Palanisamy	73	91005		*			*		1	1	0	2	1	1	1	7	17		0.2
14	Vetrivel	52	91086	*				*		0	0	0	0	0	1	0	1	18		0.8
15	Karuppan	69	91038		*			*		1	2	1	3	2	5	2	16	33	*	4.3
16	Seetapathi	54	92446	*					*	0	0	0	1	0	0	0	1	17		2.1
17	Kannan	60	92466		*			*		1	2	1	3	4	4	2	17	33		6.2
18	Manikkam	74	92478		*			*		1	1	1	1	2	1	0	7	28		1.9
19	Amsadoss	58	92486			*		*		0	0	0	1	1	1	0	3	18		0.1
20	Alaguraman	61	92512		*			*		0	1	1	3	3	2	1	11	28		2.6
21	Thangapandi	63	92522	*				*		0	0	0	1	1	2	0	4	17		0.5
22	Devadoss	70	93033			*			*	1	2	1	4	2	4	3	17	36	*	6
23	Chinnakalai	78	93101		*			*		1	1	1	1	1	1	0	6	18		2.1
24	Arunachalam	63	94108		*			*		0	1	0	3	3	3	2	12	28	*	1.3
25	Palani	55	95814	*					*	0	0	0	1	0	0	0	1	22		3.2

26	Kuppusamy	78	95916		*				*	1	3	1	2	4	5	2	18	29		4.7
27	Selvam	57	99703			*		*		0	2	0	3	3	3	1	12	24		0.3
28	Thangaraj	55	99884			*		*		0	0	0	0	0	0	0	0	16		0.9
29	Durai	60	99800	*				*		0	0	1	0	1	0	0	2	15		3
30	Krishnan	62	99916		*				*	2	2	1	4	3	4	3	19	26		2
31	Jesuraja	59	10222			*		*		0	1	0	1	1	1	0	4	16		2.2
32	Raju	60	10216	*				*		0	0	0	0	0	1	1	2	17		3.1
33	Seetaraman	51	10701	*				*		0	0	0	0	0	0	0	0	16		0.3
34	Kannan	58	1113	*				*		1	1	0	3	3	3	1	12	36	*	4.3
35	Krishnamoorthi	68	1120		*				*	1	2	0	2	2	3	1	11	32		4.4
36	Shanmugam	61	1189		*				*	0	0	0	1	1	0	0	2	17		0.6
37	Sankar	63	4462	*				*		2	3	1	3	3	3	2	17	28		0.6
38	Velusamy	60	4883			*		*		0	0	0	0	0	1	0	1	22		0.5
39	Arumugam	63	4832	*				*		1	2	1	3	2	4	2	15	36	*	3.9
40	Palanisamy	62	5545	*				*		0	0	0	0	1	1	1	3	19		1
41	Palsamy	70	5568		*				*	1	1	0	2	2	2	2	10	33	*	4.2
42	Balamurugan	72	8268		*				*	0	1	0	1	1	1	1	5	18		0.3
43	Ayyadurai	69	8298	*				*		0	0	0	0	0	0	1	1	17		0.6
44	Manikkam	57	9959	*				*		0	1	0	0	1	1	1	4	16		0.8
45	Madasamy	60	9889		*			*		0	0	0	0	0	0	0	0	16		1.1
46	Palanivel	63	9918		*				*	0	0	0	1	1	2	0	4	18		2.4
47	Ramu	60	9938	*					*	0	0	0	0	0	0	0	0	18		0.3
48	Chellaya	66	9899			*		*		0	1	0	3	3	3	2	12	28		0.6
49	Raju	62	10008			*		*		1	1	1	1	1	1	0	6	17		1.7
50	Karuppaiya	69	10014		*			*		0	0	0	1	0	0	0	1	17		1.1
51	Arunachalam	54	10049			*		*		0	0	0	0	0	0	0	0	15		0.1
52	Muthusamy	70	11563	*					*	0	2	0	3	3	3	1	12	26	*	4.2
53	Madasamy	62	11711	*				*		0	0	0	2	3	3	3	11	29		4.3
54	Ambalam	69	11683		*			*		0	1	0	0	0	0	0	1	17		3.1
55	Selvam	51	11881	*					*	0	0	0	0	0	0	0	0	17		0.1
56	Rajamani	53	12001		*			*		0	0	0	0	0	0	0	0	16		3.2
57	Karuppusamy	62	15101	*					*	3	3	3	3	2	4	4	22	36	*	3.9
58	Durai	77	15188	*				*		3	4	4	4	5	4	4	28	28		5.1

59	Muniyandi	60	15133			*		*		0	2	2	2	2	5	3	16	16		0.3
60	Samy	61	15201			*		*		0	0	0	0	0	0	0	0	16		0.2
61	Jafer Ali	58	18921			*	*	*		0	0	0	0	0	0	0	0	22		4.1
62	Natchiyambalam	69	18998		*		*	*		1	2	0	0	3	3	4	13	28		3.6
63	Muthu	72	18455			*			*	0	1	0	0	0	1	0	2	21		3.1
64	Sivaramakrishnan	54	20492	*				*		0	0	0	0	0	0	0	0	18		0.6
65	Mookan	78	20522	*					*	1	1	0	1	2	3	2	10	24		0.9
66	Rajendran	64	54470		*			*		0	0	0	0	1	1	0	2	16		0.5
67	Samayadurai	65	58510	*				*		0	0	0	0	0	0	0	0	16		0.3
68	Mayandi	72	55281	*				*		0	1	0	1	1	1	1	5	17		0.1
69	Maruthu	76	57321		*			*		1	2	1	1	3	3	2	13	32		1.2
70	Raman	55	57330	*					*	0	0	0	0	0	0	0	0	16		3.3
71	Kannan	51	57418		*			*		0	0	0	0	0	0	0	0	14		2.1
72	Aandi	58	58383		*				*	0	1	0	1	0	1	0	3	17		0.9
73	Jesuraja	63	58630		*			*		1	2	1	1	2	2	1	10	25	*	4.9
74	Anwar Pasha	60	58644		*				*	0	0	0	0	1	1	0	2	24		0.6
75	Velu	66	58659	*					*	0	0	0	0	0	0	0	0	17		0.9
76	Pandi	68	58708	*				*		0	0	0	0	0	0	0	0	18		1.9
77	Ponnuchami	69	60019			*		*		0	1	1	2	2	2	2	10	28		3.2
78	Arumugam	71	60083		*		*	*		1	1	1	1	1	1	1	7	16		3
79	Palani	66	61191	*					*	0	0	0	0	0	0	0	0	16		0.8
80	Karuppan	52	61837	*					*	0	0	0	0	0	0	0	0	16		0.1
81	Karthikeyan	57	61438			*		*		0	0	0	0	0	0	1	1	17		0.2
82	Sami	59	62921		*			*		0	0	0	0	0	1	1	2	18		0.1
83	Kizhavan	71	62983		*				*	1	2	1	3	3	3	4	17	32	*	5.6
84	Chinnakalai	65	62953		*			*		0	0	0	0	1	3	3	7	16		0.5
85	Karuppaiah	74	64963		*		*	*		1	2	1	2	2	2	2	12	28		3.1
86	Palani	78	65533	*				*		0	0	1	3	3	3	3	13	28		3.9
87	Murugan	58	65601		*			*		0	0	0	0	0	0	0	0	17		0.3
88	Raja	62	65608	*				*		0	0	0	0	1	0	1	2	16		1.3
89	Appu	64	66633		*				*	1	1	0	1	2	2	2	9	30		5.1
90	Karuppan	66	66711	*				*		0	1	0	0	0	0	0	1	16		4.3
91	Karmegam	55	67001	*			*	*		0	0	0	0	0	0	0	0	18		0.2

92	Pandi	65	67013			*		*		0	2	0	2	4	3	3	14	35	*	3.3
93	Vadivel	67	67038		*			*		1	1	1	1	1	1	1	7	15		3.6
94	Rajendran	68	67059		*				*	0	0	0	0	0	0	0	0	16		2.6
95	Andisamy	60	67383	*					*	0	0	0	0	0	0	1	1	15		2.4
96	Murugan	55	70081		*			*		0	0	0	1	0	1	0	2	15		0.3
97	Rajan	53	73183	*				*		0	0	0	0	0	0	0	0	16		0.9
98	Thirumalai	56	74101			*		*		0	1	0	0	0	0	0	1	18		1.9
99	Arumugam	58	76280			*		*		0	0	0	0	0	0	1	1	17		0.2
100	Duraipandi	65	76306		*				*	1	2	1	2	4	4	1	15	27		4.1
101	Pandi	59	78801	*				*		0	0	0	0	0	0	0	0	24		3.9
102	Sundararajan	53	79113		*			*		0	0	0	0	0	0	0	0	19		0.3
103	Gurusamy	52	79233			*		*		0	0	0	0	0	0	0	0	18		0.6
104	Chinnan	71	80413	*					*	2	2	1	2	3	3	4	17	38	*	6.1
105	Nallendrababu	60	80451			*		*		1	2	1	2	2	2	3	13	28		1.6
106	Kalailingam	78	80501			*		*		1	2	2	2	3	3	1	14	28		1.2
107	Alagesan	65	80523	*				*		0	0	0	0	0	0	0	0	17		3
108	Ramuthevar	51	80557		*				*	0	0	0	0	0	0	0	0	17		0.3
109	Ayyavu	67	83588	*					*	0	1	1	2	2	3	0	9	18		0.6
110	Muthu	60	83838			*		*		0	0	0	0	0	0	0	0	18		4.1
111	Selvam	63	91300		*				*	1	0	0	0	0	1	1	3	32		3.4
112	Lakshmanan	70	91510	*					*	1	2	1	0	4	3	2	13	28		4.6
113	Seethapathi	56	91683	*				*		0	0	0	0	0	0	0	0	18		0.9
114	Devadoss	62	92004		*			*		0	1	0	0	0	0	1	2	17		0.6
115	Krishnan	51	94183			*			*	0	0	0	0	0	0	0	0	16		0.4
116	Velusamy	75	94204	*				*		1	1	0	0	0	2	1	5	22	*	0.4
117	Sakthivel	56	94381		*				*	0	0	0	0	0	0	0	0	16		3.9
118	Sakkaraithevar	85	94386	*				*		1	2	2	3	3	3	3	17	29	*	2.1
119	Ramar	62	97101		*			*		0	0	0	0	0	0	0	0	18		1
120	Dhanabalasingam	75	97286		*			*		0	2	1	3	2	3	1	12	26		4.1
121	Murugan	62	97212	*				*		0	0	0	0	0	1	0	1	17		3.2
122	Aandi	60	381	*				*		0	0	0	0	0	0	0	0	16		3
123	Gurusamy	59	508			*		*		0	0	0	0	0	0	1	1	16		1
124	Sikkender	52	1825	*				*		0	0	0	0	0	0	0	0	15		1

125	Pandi	60	5039	*				*		0	1	1	2	2	2	1	9	24		0.9
126	Mani	56	7929	*					*	0	0	0	0	0	0	0	0	16		0.6
127	Subbu	52	8061	*	*				*	0	0	0	0	0	0	0	0	16		0.5
128	Ammasi	60	9781			*			*	0	0	0	0	0	1	1	2	17		1.2
129	Ramu	68	9798		*			*		1	1	0	1	2	2	1	8	32		4.3
130	Vanniyaperumal	51	20101		*			*		0	0	0	0	0	0	0	0	15		3.3
131	Thangapandi	58	20118		*			*		0	0	0	0	0	0	0	0	16		0.2
132	Rasu	65	20645			*			*	0	0	0	0	0	1	1	2	16		0.3
133	Chinnappan	70	22328			*	*	*		1	2	1	1	2	2	1	10	15		0.3
134	Aandi	74	22338		*			*		2	2	1	2	3	4	3	17	36	*	5.1
135	Kannan	60	26890	*				*		1	1	0	0	0	1	0	3	17		0.9
136	Raja	52	30340	*				*		0	0	0	0	0	0	0	0	18		0.6
137	Adaikan	65	35203			*		*		0	1	0	0	0	1	0	2	17		0.6
138	Subramaniyan	55	36882			*		*		0	0	0	0	0	0	0	0	17		0.3
139	Muthukalai	65	38511		*			*		0	2	0	0	3	3	3	11	26		1.6
140	Velusamy	67	40101			*		*		0	1	0	0	0	0	0	1	17		1.9
141	Manimaran	55	40118	*				*		0	0	0	0	0	0	0	0	19		0.9
142	Hariram	54	57883		*				*	0	1	0	0	0	0	0	1	19		0.3
143	Adaikan	68	62182		*			*		1	2	2	1	1	1	1	9	24		4.3
144	Sundararajan	52	62291	*	*				*	0	0	0	0	0	0	0	0	16		3.2
145	Arumugam	51	92913	*					*	0	0	0	0	0	0	0	0	15		3.3
146	Palanisamy	64	63011			*		*		1	1	0	1	2	2	1	8	26		3.9
147	Gurusamy	55	64745			*		*		0	0	0	0	0	0	0	0	19		0.6
148	Pandi	56	65639	*				*		0	0	0	0	0	0	0	0	21		3.2
149	Raja	53	65990	*				*		0	0	0	0	0	0	0	0	16		0.3
150	Amavasi	69	69512		*		*	*		1	2	2	1	1	2	2	11	33	*	4.2

MASTER CHART - CONTROLS

S.No.	Name	A	IP NO	Diagnosis	International Prostate Symptom Score	Pr.V	PRV	PSA

					Ic.Em	Fr.	In.Em	Urg.	Wk.St.	Str.	Noct.	IPSS			n/ml
1	Pitchai	70	77286	Diabetic Foot	2	2	1	2	3	3	1	14	33		4.2
2	Ponnusamy	64	77292	Hemorrhoids	0	0	0	0	0	0	1	1	17		2.3
3	Mariappan	56	78112	Hydrocele	0	0	0	0	0	0	0	0	18		2.8
4	Manikkam	72	79510	Ca.Rectum	1	0	0	2	0	0	0	3	17		2.9
5	Ayyavu	81	79624	Chronic Pancreatitis	1	3	1	3	4	4	2	18	36	*	3.1
6	Sakthivel	53	82195	Acute Gastritis	0	0	0	0	0	0	0	0	16		1.8
7	Selvam	61	82973	Gastric Outlet Obstruction	0	0	0	1	0	0	0	1	17		3
8	Kathiresan	60	83402	Diabetic Foot	0	0	0	0	0	1	0	1	18		3.6
9	Velmurugan	56	84110	Varicose veins	0	1	0	1	0	1	0	3	15		2.8
10	Baskaran	61	87197	Liver abscess	0	0	0	0	0	0	0	0	17		3.1
11	Prabakaran	71	88003	Fournier's Gangrene	0	1	1	0	1	0	0	3	18		1.9
12	Palani	82	90219	Acid Peptic Disease	1	0	0	0	2	2	0	5	16		1.8
13	Syed Mohammed	53	90993	Hemorrhoids	2	2	2	3	3	3	4	19	37	*	5.1
14	Balanayagam	61	91218	Ca.Stomach	0	0	0	0	0	0	0	0	16		2.3
15	Natchimuthu	69	91784	Malignant melanoma	0	1	0	0	0	1	0	2	21		4.2
16	Periyakaruppan	71	92763	Fissure in ano	1	2	1	3	3	3	2	15	28		4.5
17	Ayyappan	83	92901	Ca.colon	1	1	1	1	1	1	0	6	26		3.6
18	Muniyandi	62	92987	Hemorrhoids	0	0	0	0	0	0	0	0	18		3.2
19	Palpandi	65	93107	POVD	0	1	0	0	0	0	0	1	17		2.8
20	Kannan	56	93525	Cellulitis	0	0	0	0	0	0	0	0	17		1.9
21	Sudalaimuthu	59	93746	Diabetic Foot	0	0	0	0	0	0	1	1	18		1.2
22	Ammavasi	78	94399	Lipoma back	1	1	2	1	3	3	4	15	22		4.9
23	Chinnakaruppan	61	94865	Fissure in ano	0	0	0	0	0	0	0	0	22		3.7
24	Natchiyappan	63	95717	Hemorrhoids	0	2	1	2	2	2	1	10	24		6.1
25	Baskaran	65	95886	Ileocaecal TB	0	0	0	0	0	0	0	0	17		1.9
26	Namasivayam	68	96392	Diabetic Foot	0	1	0	0	0	1	0	2	18		3.7

27	Sundareshwaran	71	96801	Hemorrhoids	0	0	0	0	0	0	0	0	17		2
28	Rathinam	58	97889	Cellulitis	0	0	0	0	0	0	1	1	18		4.3
29	Alagappan	64	98241	Obstructive Jaundice	0	0	0	0	0	1	0	1	19		5.2
30	Kulandaivel	62	98369	Ca.Penis	1	2	1	3	3	3	4	17	36	*	6.1
31	Saravanamuthu	63	106231	Hemorrhoids	0	0	0	0	1	1	0	2	18		2.5
32	Arivuselvam	58	102182	Diabetic Foot	0	0	0	0	0	0	0	0	19		1.6
33	Manickam	71	102014	Varicose veins	0	0	0	0	0	0	0	0	23		4.1
34	Sudalaimuthu	78	1217	Fissure in ano	1	0	0	0	0	0	1	2	19		0.9
35	Sivankalai	64	1281	Gastric Outlet Obstruction	0	0	0	0	0	0	0	0	19		3.3
36	Karuppaih	59	1385	Cellulitis	0	0	0	2	0	0	1	3	23		2.2
37	Hassen khan	62	4432	Hemorrhoids	2	2	1	3	3	4	2	17	32	*	7.3
38	Nagan	58	4483	POVD	0	0	0	0	1	1	0	2	18		2.2
39	Krishnamoorthy	64	4842	Cellulitis	0	1	0	2	0	0	0	3	16		2.3
40	Karuppaih	73	5551	Diabetic Foot	1	3	3	3	3	4	3	20	38	*	5.3
41	Sudalaimuthu	63	5597	Diabetic Foot	0	0	0	0	1	1	0	2	22		4.6
42	Ramar	67	8061	Ca.Penis	0	1	0	0	0	1	1	3	18		0.8
43	Ramaiah	59	8088	Acid Peptic Disease	0	0	0	0	0	0	0	0	16		1.4
44	Ramu	73	9781	Cellulitis	2	2	1	2	3	3	1	14	27		5.5
45	chellaiah	77	9704	Hemorrhoids	1	1	1	2	2	2	1	10	28		6.1
46	Anbazhagan	68	9981	Liver abscess	0	0	0	0	0	0	0	0	17		0.9
47	Aruljesudason	61	9920	Hemorrhoids	1	2	1	3	4	3	2	16	32	*	3.2
48	Kannabiran	59	9916	Fournier's Gangrene	0	0	0	0	1	1	1	3	18		3.6
49	Muthusamy	72	10112	Fistula in ano	1	3	0	0	0	2	3	9	33		4.7
50	Thangarsu	60	10203	Cellulitis	0	1	0	0	1	1	0	3	17		0.6
51	Arasan	59	11638	Diabetic Foot	0	0	0	0	0	0	0	0	22		4.9
52	Pandidurai	58	12429	Ca.Stomach	0	0	1	0	1	1	1	0	16		9
53	Surendran	63	12873	Hemorrhoids	1	1	1	1	0	1	0	5	18		1.2
54	Joseph	67	13108	Cellulitis	0	0	0	0	0	1	1	2	18		1.3
55	Balamurugan	71	13910	Varicose veins	0	1	1	1	1	0	0	4	32		1.7
56	Aandiyappan	72	14539	Diabetic Foot	1	3	1	4	4	4	3	20	36	*	5.2

57	Nagendran	59	14800	Lipoma back	0	0	0	0	0	0	0	0	18		1.3
58	Thirumalai	60	15006	Obstructive Jaundice	0	1	0	0	0	1	0	2	17		1.5
59	Anwar shahid	61	15123	Acid Peptic Disease	0	0	0	0	0	1	1	2	18		1.9
60	Chellapandi	68	15293	Hydrocele	1	1	0	0	0	1	0	3	16		0.5
61	Baranitharan	60	16819	Hemorrhoids	0	0	0	0	0	0	1	1	18		0.6
62	Basha ali	75	17801	Diabetic Foot	0	0	0	2	3	3	0	8	32		4.1
63	Mayandi	59	17912	Ca.Rectum	0	0	0	1	0	0	1	2	17		0.1
64	Pandidurai	61	18536	Cellulitis	1	0	0	0	0	0	0	1	18		0.8
65	Ariularasan	80	18773	Diabetic Foot	0	0	0	0	0	0	3	3	28	*	4.1
66	Boominathan	61	19214	Acid Peptic Disease	0	0	0	0	0	1	1	2	18		0.6
67	Raja	54	19348	Cellulitis	1	2	1	2	3	3	3	15	24		4.2
68	Madasamy	62	19876	Cellulitis	0	0	0	0	1	0	1	2	18		0.9
69	Velmurugan	71	20312	Varicose veins	0	1	0	0	1	1	0	3	19		0.8
70	Annamalai	69	20610	Hemorrhoids	0	1	0	0	1	1	1	4	28		1.5
71	Chandrasekaran	62	28312	Acid Peptic Disease	0	1	0	1	1	0	0	3	19		3.9
72	Jesuraja	73	29103	Acid Peptic Disease	0	0	0	1	1	1	1	4	18		2.1
73	Baskaran	60	30943	Hemorrhoids	1	2	0	1	2	3	4	13	36	*	5.5
74	Syed Ibrahim	81	38412	Cellulitis	0	1	0	1	1	1	2	6	32		0.6
75	Sivaraman	71	40198	Diabetic Foot	0	1	0	0	0	0	0	1	18		2.2
76	Kannan	63	43619	Hemorrhoids	0	0	0	0	0	3	2	5	22		3
77	Palani	72	45900	Ca.Penis	0	0	0	0	0	0	0	0	18		0.6
78	Ramakrishnan	61	48665	Diabetic Foot	0	0	0	0	1	1	0	2	17		0.7
79	Chandran	60	50012	Diabetic Foot	0	1	0	0	1	1	1	4	18		1.1
80	Palpandi	59	53118	Cellulitis	0	0	0	0	0	0	1	1	17		2.5
81	Ammavasi	64	55809	Acid Peptic Disease	0	1	0	1	1	1	1	5	24		3.6
82	Ulaganathan	71	56962	Fissure in ano	1	2	1	4	3	3	3	17	18		3.4
83	Senthilvel	62	57361	Liver abscess	0	1	0	0	1	1	0	3	18		0.8
84	Karuppaih	83	62646	Diabetic Foot	3	3	2	4	5	5	5	27	38		5.1
85	Selvaraj	58	63129	Cellulitis	1	0	1	1	0	1	1	5	17		0.9
86	Anbazhagan	65	64080	Lipoma	0	0	0	1	0	1	0	2	16		0.3

87	Rajasekaran	54	65274	Ca.Stomach	0	0	0	1	1	1	0	3	17		0.1
88	Karuppan	56	66918	Hemorrhoids	0	1	1	1	0	0	0	3	18		1
89	Chinnasamy	68	67205	Hemorrhoids	0	0	0	0	0	0	0	0	18		1.5
90	Venkateswaran	70	67313	Gastric Outlet Obstruction	0	0	1	0	1	1	1	4	19		1.3
91	Ambalam	70	68516	Ca.colon	0	1	0	0	0	0	2	3	28		3.9
92	Pasha	72	68565	Acid Peptic Disease	0	1	0	1	1	1	1	5	19		3.1
93	Mayilvahanan	61	69129	Cellulitis	0	0	0	0	0	0	0	0	17		0.2
94	Pitchai	65	71193	Diabetic Foot	1	2	1	3	4	4	5	20	36	*	6.1
95	Muniyandi	63	72456	Hemorrhoids	0	0	0	0	1	1	0	2	17		0.3
96	Annadurai	58	72964	Chronic Pancreatitis	0	0	0	1	0	0	0	1	18		0.5
97	Manickam	53	73291	Basal Cell Carcinoma	2	3	2	0	4	4	4	19	24	*	1.6
98	Kathiravan	61	74643	Cellulitis	0	0	0	0	1	1	1	3	18		1.1
99	Ramachandran	65	74998	Hemorrhoids	0	1	0	1	0	1	1	4	17		2
100	Natarajan	70	75109	Acid Peptic Disease	0	0	0	2	2	1	0	5	32		2.1
101	Shanmugam	71	75325	Obstructive Jaundice	1	0	0	1	1	0	1	4	24		2.6
102	Arumugam	60	76543	Hydrocele	1	0	0	1	1	0	0	3	18		2.4
103	Palanivel	62	79100	Ca.Penis	1	0	1	1	0	1	1	5	18		2.9
104	Ponnan	68	79856	Hemorrhoids	1	1	1	2	2	4	4	15	32		4.3
105	Periyakaruppan	72	80214	POVD	2	2	1	3	3	4	1	16	29		4.1
106	Sundaram	61	82315	Diabetic Foot	1	1	0	1	1	2		6	24		1.1
107	chellaiah	70	82963	POVD	2	4	4	3	4	5	4	26	38	*	5.1
108	Raja	51	83516	Acid Peptic Disease	1	0	1	1	1	1	0	5	18		3.1
109	Kumarvel	63	83600	Fournier's Gangrene	0	0	0	0	0	0	0	0	17		0.6
110	Sami	55	83622	Fistula in ano	1	1	1	1	1	2	1	8	29		3.5
111	Sakthivel	61	83642	Varicose veins	0	0	0	0	0	0	0	0	18		0.2
112	Vadivel	58	84873	Hemorrhoids	0	0	0	0	0	0	0	0	17		0.1
113	Sundaram	53	84902	Ca.Rectum	0	1	0	0	0	1	1	3	18		0.3
114	Kannan	68	85762	Gastric Outlet Obstruction	0	0	0	1	1	2	2	6	19		0.4
115	Kannabiran	71	86594	Diabetic Foot	0	0	0	0	0	0	0	0	36		0.9
116	Veluchamy	65	87258	Acute Gastritis	0	1	0	1	1	1	1	5	18		0.6

117	Paraman	60	89163	Diabetic Foot	0	1	1	0	1	1	1	5	19		0.5
118	Rathinam	61	90003	Gastric Outlet Obstruction	0	0	0	1	1	2	3	7	18		1.5
119	Pandidurai	52	92596	Acute Gastritis	0	1	1	1	2	1	2	8	24		1.9
120	**Balamurugan**	59	93860	Diabetic Foot	1	2	1	2	2	2	4	14	28	*	5
121	Kannan	57	94426	Lipoma	0	0	0	0	0	0	0	0	17		0.2
122	Palpandi	53	95482	Fissure in ano	0	1	0	1	1	0	1	4	15		0.3
123	Raja	66	96124	Diabetic Foot	1	1	1	1	1	1	1	7	19		0.3
124	Aasirvatham	57	2134	Cellulitis	0	1	0	0	0	1	3	5	19		3
125	Parameswaran	71	4947	Diabetic Foot	2	2	1	4	2	4	3	17	16		0.1
126	Mayandi	69	5124	Hemorrhoids	0	1	0	1	0	0	0	2	18		0.5
127	Alagusamy	68	5897	Hydrocele	1	1	2	1	1	1	2	9	28		2.1
128	Murugan	71	6624	Varicose veins	1	2	1	2	3	3	3	15	24	*	3.1
129	Dharmendran	50	6756	Gastric Outlet Obstruction	0	0	0	0	0	0	0	0	18		0.5
130	Gnanasundaram	51	7123	Fissure in ano	0	0	0	0	0	1	1	2	15		0.6
131	Mohanan	64	8164	Hemorrhoids	0	1	1	0	0	1	0	3	18		0.7
132	Ganesan	59	8349	Acid Peptic Disease	1	0	0	0	1	0	1	3	17		0.5
133	Subramani	69	8972	Varicose veins	1	0	1	1	0	1	1	5	18		1.6
134	Govindarajan	72	9964	Diabetic Foot	1	2	1	3	3	5	4	19	34	*	7
135	Sankar	54	10294	POVD	0	1	0	1	1	0	1	4	16		3
136	Pandi	63	14296	Fissure in ano	1	1	1	2	2	3	1	11	28		2.3
137	Aalappan	52	15812	Chronic Pancreatitis	0	0	1	0	1	1	0	3	16		2.6
138	Ayadurai	51	19115	Ca.Stomach	1	1	0	1	0	1	0	4	18		3.1
139	Seetharaman	72	22548	Hemorrhoids	2	2	2	4	5	5	5	25	38	*	4.3
140	Narendaran	58	27519	Cellulitis	0	0	0	0	0	0	0	0	18		0.3
141	Aandi	56	31329	Cellulitis	0	1	1	0	1	2	0	5	18		0.3
142	Manoharan	53	37258	Hemorrhoids	1	0	1	1	0	1	0	4	17		0.2
143	Sundaram	56	38629	Acid Peptic Disease	1	0	0	0	1	1	0	3	17		0.9
144	Veerapandi	59	44326	Ileocaecal TB	0	0	0	0	0	0	0	0	18		0.4
145	Jesudason	58	47293	Hemorrhoids	0	1	0	0	0	0	1	2	15		1.1
146	Samy	60	55921	Ca.Rectum	2	2	2	3	4	3	1	17	27		1.9

147	Muthupandi	55	59664	Hemorrhoids	0	1	0	1	0	0	0	2	16		3.9
148	Sadik pasha	52	60215	Fissure in ano	1	0	1	1	0	1	1	5	19		3.6
149	Pandi	61	63816	Acid Peptic Disease	1	2	1	4	4	4	1	17	38		6.1
150	Alagappan	51	63994	Diabetic Foot	0	0	1	1	1	0	1	4	15		3.1